Pharmacology: Respiratory and Endocrine

Question 1 of 76

Which of the following drugs decreases plasma-theophylline levels:

a Erythromycin

b Allopurinol

c Carbamazepine

d Fluconazole

e Verapamil

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Pharmacology: Respiratory and Endocrine



Question 1 of 76

Which of the following drugs decreases plasma-theophylline levels:

a) **Erythromycin** ✖
b) Allopurinol
c) Carbamazepine ✔
d) Fluconazole
e) Verapamil

Answer

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

Question 2 of 76

Regarding the use of oxygen therapy in the management of acute asthma in adults which of the following statements is CORRECT:

a All patients with severe acute asthma should be given oxygen therapy regardless of oxygen saturations.

b Hypoxaemic patients should be given oxygen therapy to maintain saturations > 98%.

c Patients should be given oxygen therapy to maintain saturations 94 – 98%.

d Patients should not commence oxygen therapy until SpO2 has been monitored.

e All patients with SpO2 < 98% should be given oxygen therapy.

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Pharmacology: Respiratory and Endocrine

Question 2 of 76

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a)
All patients with severe acute asthma should be given oxygen therapy regardless of oxygen saturations.
b) Hypoxaemic patients should be given oxygen therapy to maintain saturations > 98%.
c) Patients should be given oxygen therapy to maintain saturations 94 – 98%.
d) Patients should not commence oxygen therapy until SpO2 has been monitored.
e) All patients with SpO2 < 98% should be given oxygen therapy.

Answer

The BTS/SIGN guidelines state: 'Give controlled supplementary oxygen to all hypoxaemic patients with acute severe asthma to maintain an SpO2 level of 94–98%. Do not delay oxygen administration in the absence of pulse oximetry but commence monitoring of SaO2 as soon as it becomes available.' Oxygen saturation (SpO2) measured by pulse oximetry determines the adequacy of oxygen therapy and the need for arterial blood gas (ABG) measurement. Patients with SpO2 < 92% or other features of life-threatening asthma require ABG measurement.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

Question 3 of 76

Which of the following clinical features would characterise an asthma attack in an adult as severe:

a PEF 55% of best/predicted

b RR 24 breaths/minute

c BP 110/70

d HR 115 bpm

e Increasing symptoms

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Pharmacology: Respiratory and Endocrine

Question 3 of 76

Which of the following clinical features would characterise an asthma attack in an adult as severe:

a) PEF 55% of best/predicted ✗
b) RR 24 breaths/minute
c) BP 110/70
d) HR 115 bpm ✓
e) Increasing symptoms

Answer

Moderate asthma is defined as:

- Increasing symptoms
- PEFR > 50 – 75% of best or predicted
- No features of acute severe asthma

Severe asthma is defined as any one of the following:

- PEFR 33 – 50% of best or predicted
- Respiratory rate ≥ 25 breaths/minute
- Heart rate ≥ 110 beats/minute
- Inability to complete sentences in one breath

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

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Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

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  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
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- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
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Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

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Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

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Pharmacology: Respiratory and Endocrine

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Which of the following is a common side effect of salbutamol:

a Ankle oedema

b Hyperkalaemia

c Bradycardia

d Weight gain

e Tremor

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Which of the following is a common side effect of salbutamol:

a) Ankle oedema
b) Hyperkalaemia
c) Bradycardia
d) Weight gain
e) **Tremor** ✔

Answer

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
- Hypokalaemia
- Cardiac arrhythmia and paradoxical bronchospasm (rare)
- Acute angle-closure glaucoma
- QT-interval prolongation

Notes

Selective beta-2 agonists act directly on beta-2 receptors, causing smooth muscle relaxation and dilation of the airways.

Mild to moderate symptoms of asthma respond rapidly to the inhalation of a selective short-acting beta2 agonist such as salbutamol or terbutaline sulfate. Short-acting beta-2 agonists have a rapid onset of action (15 minutes) and their effects last for up to 4 hours. Salbutamol or terbutaline sulfate can be given intravenously for severe or life-threatening acute asthma; patients should be carefully monitored and the dose adjusted according to response and heart rate.

Short acting beta-2 agonists are used for immediate relief of asthma symptoms, while some long-acting β2 agonists (e.g. salmeterol) are added to an inhaled corticosteroid in patients requiring prophylactic treatment.

Cautions

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Interactions

Hypokalaemia may be potentiated by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics. This in turn may predispose to toxicity in patients taking digoxin.

Side effects

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
- Hypokalaemia
- Cardiac arrhythmia and paradoxical bronchospasm (rare)
- Acute angle-closure glaucoma
- QT-interval prolongation

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Pharmacology: Respiratory and Endocrine

Question 5 of 76

A 10 year old patient, with a known allergy to nuts, is brought into ED with wheezing and difficulty in breathing, after eating cake at a birthday party. You suspect anaphylaxis. What is the appropriate dose of adrenaline to administer to this patient:

a 50 milligrams intramuscularly

b 5 milligrams intramuscularly

c 500 micrograms intramuscularly

d 300 micrograms intramuscularly

e 150 micrograms intramuscularly

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Pharmacology: Respiratory and Endocrine

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a) **50 milligrams intramuscularly** ✗
b) 5 milligrams intramuscularly
c) 500 micrograms intramuscularly
d) 300 micrograms intramuscularly ✓
e) 150 micrograms intramuscularly

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Answer

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

Question 6 of 76

Ipratropium bromide should be used with caution in patients with which of the following conditions:

a Convulsive disorders

b Prostatic hyperplasia

c Hypokalaemia

d Diabetes mellitus

e Hyperthyroidism

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Pharmacology: Respiratory and Endocrine



Question 6 of 76

Ipratropium bromide should be used with caution in patients with which of the following conditions:

a) Convulsive disorders
b) **Prostatic hyperplasia** ✔
c) Hypokalaemia
d) Diabetes mellitus
e) Hyperthyroidism

Answer

Ipratropium bromide should be used with caution in:

- Men with prostatic hyperplasia and bladder-outflow obstruction (worsened urinary retention has been reported in elderly men)
- People with chronic kidney disease (CKD) stages 3 and above (because of the risk of drug toxicity)
- People with angle-closure glaucoma (nebulised mist of antimuscarinic drugs can precipitate or worsen acute angle-closure glaucoma)

Notes

Ipratropium bromide, a short-acting antimuscarinic bronchodilator, causes bronchodilation by blocking the cholinergic nerves in the airways.

Ipratropium bromide can provide short-term relief in chronic asthma, but short-acting beta-2 agonists act more quickly and are preferred. Ipratropium bromide by nebulisation can be added to other standard treatment in life-threatening asthma or if acute asthma fails to improve with standard therapy.

The aerosol inhalation of ipratropium bromide may be used for short-term relief in mild COPD in patients who are not using a long-acting antimuscarinic drug. Its maximal effect occurs 30 – 60 minutes after use; its duration of action is 3 to 6 hours and bronchodilation can usually be maintained with treatment 3 times a day.

Cautions

Ipratropium bromide should be used with caution in:

- Men with prostatic hyperplasia and bladder-outflow obstruction (worsened urinary retention has been reported in elderly men)
- People with chronic kidney disease (CKD) stages 3 and above (because of the risk of drug toxicity)
- People with angle-closure glaucoma (nebulised mist of antimuscarinic drugs can precipitate or worsen acute angle-closure glaucoma)

Interactions

There are no important drug interactions with inhaled muscarinic antagonists.

Side effects

Inhaled antimuscarinics are generally well tolerated as they are poorly absorbed systemically.

Their adverse effects include:

- Dry mouth and abnormal taste in the mouth
- Nasal congestion and dryness of nasal mucosa
- Acute angle-closure glaucoma (reported in people on nebulised ipratropium)

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Pharmacology: Respiratory and Endocrine

Question 7 of 76

Regarding theophylline, which of the following statements is CORRECT:

a Theophylline is an antimuscarinic bronchodilator.

b Theophylline is effective in acute exacerbations of COPD.

c Theophylline is predominantly cleared by the kidneys.

d Clearance of theophylline is increased in smokers.

e Theophylline may cause severe hyperkalaemia and potassium levels should be monitored.

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Pharmacology: Respiratory and Endocrine

Question 7 of 76

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a) **Theophylline is an antimuscarinic bronchodilator.** ✖
b) Theophylline is effective in acute exacerbations of COPD.
c) Theophylline is predominantly cleared by the kidneys.
d) Clearance of theophylline is increased in smokers. ✔
e) Theophylline may cause severe hyperkalaemia and potassium levels should be monitored.

Answer

The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.
Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP). Theophylline is metabolised in the liver, and has a narrow therapeutic index. It is not generally effective in exacerbations of chronic obstructive pulmonary disease. Severe hypokalaemia may develop in severe asthma due concomitant treatment with beta-agonists, theophylline and its derivatives, corticosteroids, and diuretics.

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

Question 8 of 76

Which of the following is NOT a typical side effect of theophylline:

a Hypokalaemia

b Headache

c Bradycardia

d Convulsions

e Gastric irritation

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Pharmacology: Respiratory and Endocrine



Question 8 of 76

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Answer

Side effects include:

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Pharmacology: Respiratory and Endocrine

Question 9 of 76

What is the mechanism of action of cetirizine:

a H1-receptor antagonist

b Histamine agonist

c H2-receptor antagonist

d Leukotriene receptor antagonist

e Muscarinic antagonist

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Question 9 of 76

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a) **H1-receptor antagonist** ✔
b) Histamine agonist
c) H2-receptor antagonist
d) Leukotriene receptor antagonist
e) Muscarinic antagonist

Answer

Cetirizine is a competitive inhibitor at the H1-receptor (an anti-histamine).

Notes

Antihistamines are competitive inhibitors at the H1-receptor (in contrast to H2 receptor antagonists used to decrease gastric acid secretion). They act to relax histamine-induced bronchoconstriction, block the vasodilator effect of histamine, inhibit histamine-induced increases in capillary permeability and block mucus secretion and sensory nerve stimulation.

Histamine (H1)-receptor antagonists are well absorbed after oral administration. The effects of these agents are usually seen in 30 minutes (with maximal effects at 1 – 2 h); the duration of action is 3 – 8 hours for first-generation compounds and 3 – 24 hours for second-generation compounds.

H1-receptor antagonists are metabolised in the liver; many induce microsomal enzymes and alter their own metabolism and that of other drugs.

Indications

- Allergic rhinitis and conjunctivitis
- Urticarial rashes, pruritus, insect bites and stings
- Angioedema
- Anaphylaxis (second line adjunct to adrenaline)
- Nausea/vomiting and prevention of motion sickness
- Insomnia

Chlorphenamine

All older antihistamines cause sedation but alimemazine tartrate and promethazine may be more sedating whereas chlorphenamine maleate and cyclizine may be less so. This sedating activity is sometimes used to manage the pruritus associated with some allergies or used to manage occasional insomnia. There is little evidence that any one of the older, 'sedating' antihistamines is superior to another and patients vary widely in their response.

Cetirizine

The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent.

Cautions

Antihistamines should usually be avoided in acute porphyrias (although some antihistamines are thought to be safe).

Antihistamines should be used with caution in epilepsy, prostatic hypertrophy, urinary hypertension, hepatic impairment and susceptibility to angle-closure glaucoma.

Side effects (significantly reduced with second-generation agents)

Elderly patients and children are more susceptible to side effects.

Common side effects of antihistamines may include:

- Anticholinergic effects (blurred vision, dry mouth, urinary retention)
- Headache
- Gastrointestinal disturbances
- Psychomotor impairment (sedation, dizziness and loss of appetite)

Drowsiness may affect performance of skilled tasks (e.g. cycling or driving); sedating effects are enhanced by alcohol and opioid analgesics.

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Pharmacology: Respiratory and Endocrine

Question 10 of 76

Which of the following is NOT a mineralocorticoid effect of corticosteroids:

a Hypertension

b Oedema

c Potassium loss

d Calcium loss

e Hyperglycaemia

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Pharmacology: Respiratory and Endocrine



Question 10 of 76

Which of the following is NOT a mineralocorticoid effect of corticosteroids:

a) Hypertension
b) Oedema
c) Potassium loss
d) **Calcium loss** ✖
e) Hyperglycaemia ✔

Answer

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Notes

In comparing the relative potencies of corticosteroids in terms of their anti-inflammatory effects, it is worth noting that high glucocorticoid activity in itself is of no advantage unless it is accompanied by relatively low mineralocorticoid activity.

Anti-inflammatory potency

Equivalent anti-inflammatory doses of corticosteroids:

Prednisolone 5 mg = Dexamethasone 750 micrograms = Hydrocortisone 20 mg

This does not take into account their mineralocorticoid effects, nor their duration of action.

Side effects

Overdosage or prolonged use can exaggerate some of the normal physiological actions of corticosteroids leading to mineralocorticoid and glucocorticoid side effects. Mineralocorticoid side effects are most marked with fludrocortisone, but are significant with hydrocortisone, negligible with the high potency glucocorticoids, betamethasone and dexamethasone, and occur only slightly with methylprednisolone and prednisolone.

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Side effects can be minimised by using the lowest effective dose for the minimum period possible. The suppressive action of a corticosteroid on cortisol secretion is least when it is given as a single dose in the morning. Whenever possible local treatment with creams, intra-articular injections, inhalations, eye drops, or enemas should be used in preference to systemic treatment.

Adrenal suppression

During prolonged therapy with corticosteroids, particularly with systemic use, adrenal atrophy develops and can persist for years after stopping. Abrupt withdrawal after a prolonged period can lead to acute adrenal insufficiency, hypotension, or death. Patients removed from long-term glucocorticoid therapy must be weaned off the drug over several days, using progressively lower doses to allow recovery of adrenal responsiveness.

To compensate for a diminished adrenocortical response caused by prolonged corticosteroid treatment, any significant intercurrent illness, trauma, or surgical procedure requires a temporary increase in corticosteroid dose, or if already stopped, a temporary reintroduction of corticosteroid treatment.

Patients on long-term corticosteroid treatment should carry a steroid treatment card which gives guidance on minimising risk and provides details of prescriber, drug, dosage and duration of treatment.

Infections

Prolonged courses of corticosteroids increase susceptibility to infections and severity of infections; clinical presentation of infections may also be atypical.

Unless they have had chickenpox, patients receiving oral or parenteral corticosteroids for purposes other than replacement should be regarded as being at risk of severe chickenpox. Manifestations of fulminant illness include pneumonia, hepatitis and disseminated intravascular coagulation; rash is not necessarily a prominent feature. Passive immunisation with varicella–zoster immunoglobulin is needed for exposed non–immune patients receiving systemic corticosteroids or for those who have used them within the previous 3 months. Confirmed chickenpox warrants specialist care and urgent treatment. Corticosteroids should not be stopped and dosage may need to be increased.

Patients taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Hydrocortisone

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Prednisolone

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Dexamethasone

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is sufficient to inhibit corticotropin secretion for 24 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

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Pharmacology: Respiratory and Endocrine

Question 11 of 76

Which of the following types of food is most commonly implicated in anaphylactic reactions:

a Eggs

b Milk

c Nuts

d Shellfish

e Chickpeas

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Pharmacology: Respiratory and Endocrine

Question 11 of 76

Which of the following types of food is most commonly implicated in anaphylactic reactions:

a) Eggs
b) Milk
c) **Nuts** ✔
d) Shellfish
e) Chickpeas

Answer

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic. Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

Question 12 of 76

Which of the following drug classes is most commonly implicated in anaphylactic reactions:

a Thrombolytic drugs

b ACE inhibitors

c Local anaesthetics

d NSAIDs

e Opioids

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Pharmacology: Respiratory and Endocrine



Question 12 of 76

Which of the following drug classes is most commonly implicated in anaphylactic reactions:

a) Thrombolytic drugs
b) ACE inhibitors
c) Local anaesthetics
d) **NSAIDs** ✔
e) Opioids

Answer

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic. Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

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Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

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- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

Question 13 of 76

Which of the following is NOT an indication for an antihistamine:

a Anaphylaxis

b Nausea and vomiting

c Allergic rhinitis

d Insomnia

e Gastroesophageal reflux disease

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Pharmacology: Respiratory and Endocrine



Question 13 of 76

Which of the following is NOT an indication for an antihistamine:

a) Anaphylaxis
b) Nausea and vomiting
c) Allergic rhinitis
d) Insomnia
e) **Gastroesophageal reflux disease** ✔

Answer

Antihistamines are competitive inhibitors at the H1-receptor (in contrast to H2 receptor antagonists used to decrease gastric acid secretion in gastroesophageal reflux disease).

Indications:

- Allergic rhinitis and conjunctivitis
- Urticarial rashes, pruritus, insect bites and stings
- Angioedema
- Anaphylaxis (second line adjunct to adrenaline)
- Nausea/vomiting and prevention of motion sickness
- Insomnia

Notes

Antihistamines are competitive inhibitors at the H1-receptor (in contrast to H2 receptor antagonists used to decrease gastric acid secretion). They act to relax histamine-induced bronchoconstriction, block the vasodilator effect of histamine, inhibit histamine-induced increases in capillary permeability and block mucus secretion and sensory nerve stimulation.

Histamine (H1)-receptor antagonists are well absorbed after oral administration. The effects of these agents are usually seen in 30 minutes (with maximal effects at 1 – 2 h); the duration of action is 3 – 8 hours for first-generation compounds and 3 – 24 hours for second-generation compounds.

H1-receptor antagonists are metabolised in the liver; many induce microsomal enzymes and alter their own metabolism and that of other drugs.

Indications

- Allergic rhinitis and conjunctivitis
- Urticarial rashes, pruritus, insect bites and stings
- Angioedema
- Anaphylaxis (second line adjunct to adrenaline)
- Nausea/vomiting and prevention of motion sickness
- Insomnia

Chlorphenamine

All older antihistamines cause sedation but alimemazine tartrate and promethazine may be more sedating whereas chlorphenamine maleate and cyclizine may be less so. This sedating activity is sometimes used to manage the pruritus associated with some allergies or used to manage occasional insomnia. There is little evidence that any one of the older, 'sedating' antihistamines is superior to another and patients vary widely in their response.

Cetirizine

The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent.

Cautions

Antihistamines should usually be avoided in acute porphyrias (although some antihistamines are thought to be safe).

Antihistamines should be used with caution in epilepsy, prostatic hypertrophy, urinary hypertension, hepatic impairment and susceptibility to angle-closure glaucoma.

Side effects (significantly reduced with second-generation agents)

Elderly patients and children are more susceptible to side effects.

Common side effects of antihistamines may include:

- Anticholinergic effects (blurred vision, dry mouth, urinary retention)
- Headache
- Gastrointestinal disturbances
- Psychomotor impairment (sedation, dizziness and loss of appetite)

Drowsiness may affect performance of skilled tasks (e.g. cycling or driving); sedating effects are enhanced by alcohol and opioid analgesics.

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Pharmacology: Respiratory and Endocrine

Question 14 of 76

A 19 year old student presents to ED with severe acute asthma. You prescribe a salbutamol and ipratropium bromide nebuliser. What is the most appropriate dose of salbutamol to prescribe initially for this patient:

a 10 mg every 10 minutes

b 5 mg every 15 – 30 minutes

c 2.5 mg every 10 – 20 minutes

d 0.5 mg every 20 – 30 minutes

e 0.5 mcg every 10 – 2 minutes

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Pharmacology: Respiratory and Endocrine

Question 14 of 76

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a) 10 mg every 10 minutes
b) **5 mg every 15 – 30 minutes** ✔
c) 2.5 mg every 10 – 20 minutes
d) 0.5 mg every 20 – 30 minutes
e) 0.5 mcg every 10 – 2 minutes

Answer

High-dose inhaled short-acting beta2-agonists are the first line treatment for acute asthma. A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary). The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals). Continuous nebulisation should be considered in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour). The intravenous route should be reserved for those in whom inhaled therapy cannot be used reliably.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

Question 15 of 76

Hydrocortisone would be most useful for which of the following conditions:

a Raised intracranial pressure secondary to malignancy

b Neuropathic postural hypotension

c Mineralocorticoid replacement in Addison's disease

d Long-term suppression in rheumatoid arthritis

e Emergency management of anaphylaxis

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Pharmacology: Respiratory and Endocrine



Question 15 of 76

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a) Raised intracranial pressure secondary to malignancy ✖
b) Neuropathic postural hypotension
c) Mineralocorticoid replacement in Addison's disease
d) Long-term suppression in rheumatoid arthritis
e) Emergency management of anaphylaxis ✔

Answer

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Notes

In comparing the relative potencies of corticosteroids in terms of their anti-inflammatory effects, it is worth noting that high glucocorticoid activity in itself is of no advantage unless it is accompanied by relatively low mineralocorticoid activity.

Anti-inflammatory potency

Equivalent anti-inflammatory doses of corticosteroids:

Prednisolone 5 mg = Dexamethasone 750 micrograms = Hydrocortisone 20 mg

This does not take into account their mineralocorticoid effects, nor their duration of action.

Side effects

Overdosage or prolonged use can exaggerate some of the normal physiological actions of corticosteroids leading to mineralocorticoid and glucocorticoid side effects. Mineralocorticoid side effects are most marked with fludrocortisone, but are significant with hydrocortisone, negligible with the high potency glucocorticoids, betamethasone and dexamethasone, and occur only slightly with methylprednisolone and prednisolone.

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Side effects can be minimised by using the lowest effective dose for the minimum period possible. The suppressive action of a corticosteroid on cortisol secretion is least when it is given as a single dose in the morning. Whenever possible local treatment with creams, intra-articular injections, inhalations, eye drops, or enemas should be used in preference to systemic treatment.

Adrenal suppression

During prolonged therapy with corticosteroids, particularly with systemic use, adrenal atrophy develops and can persist for years after stopping. Abrupt withdrawal after a prolonged period can lead to acute adrenal insufficiency, hypotension, or death. Patients removed from long-term glucocorticoid therapy must be weaned off the drug over several days, using progressively lower doses to allow recovery of adrenal responsiveness.

To compensate for a diminished adrenocortical response caused by prolonged corticosteroid treatment, any significant intercurrent illness, trauma, or surgical procedure requires a temporary increase in corticosteroid dose, or if already stopped, a temporary reintroduction of corticosteroid treatment.

Patients on long-term corticosteroid treatment should carry a steroid treatment card which gives guidance on minimising risk and provides details of prescriber, drug, dosage and duration of treatment.

Infections

Prolonged courses of corticosteroids increase susceptibility to infections and severity of infections; clinical presentation of infections may also be atypical.

Unless they have had chickenpox, patients receiving oral or parenteral corticosteroids for purposes other than replacement should be regarded as being at risk of severe chickenpox. Manifestations of fulminant illness include pneumonia, hepatitis and disseminated intravascular coagulation; rash is not necessarily a prominent feature. Passive immunisation with varicella–zoster immunoglobulin is needed for exposed non–immune patients receiving systemic corticosteroids or for those who have used them within the previous 3 months. Confirmed chickenpox warrants specialist care and urgent treatment. Corticosteroids should not be stopped and dosage may need to be increased.

Patients taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Hydrocortisone

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Prednisolone

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Dexamethasone

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is sufficient to inhibit corticotropin secretion for 24 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

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Pharmacology: Respiratory and Endocrine

Question 16 of 76

A 35 year old woman presents to ED with an acute asthma attack and you are asked to prescribe steroids. What is the most appropriate dose of oral prednisolone to prescribe for this patient:

a 20 mg daily

b 30 mg daily

c 40 mg daily

d 60 mg daily

e 80 mg daily

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Pharmacology: Respiratory and Endocrine

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c) 40 mg daily ✓
d) 60 mg daily
e) 80 mg daily

Answer

The BTS/SIGN guidelines state that steroids in adequate doses should be given to all patients with an acute asthma attack. Prednisolone 40 – 50 mg should be prescribed daily for at least 5 days or until recovery. Parenteral hydrocortisone (400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
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Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
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Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

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Pharmacology: Respiratory and Endocrine

Question 17 of 76

Which of the following is a common side effect of ipratropium bromide:

a Ankle oedema

b Oral candidiasis

c Bradycardia

d Dry mouth

e Tremor

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Pharmacology: Respiratory and Endocrine



Question 17 of 76

Which of the following is a common side effect of ipratropium bromide:

a) Ankle oedema
b) **Oral candidiasis** ✖
c) Bradycardia
d) Dry mouth ✔
e) Tremor

Answer

Inhaled antimuscarinics are generally well tolerated as they are poorly absorbed systemically.

Their adverse effects include:

- Dry mouth and abnormal taste in the mouth
- Nasal congestion and dryness of nasal mucosa
- Acute angle-closure glaucoma (reported in people on nebulised ipratropium)

Notes

Ipratropium bromide, a short-acting antimuscarinic bronchodilator, causes bronchodilation by blocking the cholinergic nerves in the airways.

Ipratropium bromide can provide short-term relief in chronic asthma, but short-acting beta-2 agonists act more quickly and are preferred. Ipratropium bromide by nebulisation can be added to other standard treatment in life-threatening asthma or if acute asthma fails to improve with standard therapy.

The aerosol inhalation of ipratropium bromide may be used for short-term relief in mild COPD in patients who are not using a long-acting antimuscarinic drug. Its maximal effect occurs 30 – 60 minutes after use; its duration of action is 3 to 6 hours and bronchodilation can usually be maintained with treatment 3 times a day.

Cautions

Ipratropium bromide should be used with caution in:

- Men with prostatic hyperplasia and bladder-outflow obstruction (worsened urinary retention has been reported in elderly men)
- People with chronic kidney disease (CKD) stages 3 and above (because of the risk of drug toxicity)
- People with angle-closure glaucoma (nebulised mist of antimuscarinic drugs can precipitate or worsen acute angle-closure glaucoma)

Interactions

There are no important drug interactions with inhaled muscarinic antagonists.

Side effects

Inhaled antimuscarinics are generally well tolerated as they are poorly absorbed systemically.

Their adverse effects include:

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Pharmacology: Respiratory and Endocrine

Question 18 of 76

A 19 year old student presents to ED with severe acute asthma. You prescribe a salbutamol and ipratropium bromide nebuliser. What is the most appropriate dose of ipratropium bromide to prescribe initially for this patient:

a 1 mg 4 – 6 hourly

b 0.05 mg every 2 hours

c 0.5 mg 4 – 6 hourly

d 2.5 mg 2 – 4 hourly

e 5 mg hourly

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Pharmacology: Respiratory and Endocrine

Question 18 of 76

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a) **1 mg 4 – 6 hourly** ✗
b) 0.05 mg every 2 hours
c) 0.5 mg 4 – 6 hourly ✓
d) 2.5 mg 2 – 4 hourly
e) 5 mg hourly

Answer

The BTS/SIGN guidelines state: Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
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Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

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  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
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  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
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Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

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Pharmacology: Respiratory and Endocrine

Question 19 of 76

Dexamethasone would be most useful for which of the following conditions:

a Raised intracranial pressure secondary to malignancy

b Mineralocorticoid replacement in adrenal insufficiency

c Long-term suppression in rheumatoid arthritis

d Topical use in eczema

e Emergency management of anaphylaxis

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Pharmacology: Respiratory and Endocrine



Question 19 of 76

Dexamethasone would be most useful for which of the following conditions:

a) Raised intracranial pressure secondary to malignancy ✔
b) Mineralocorticoid replacement in adrenal insufficiency
c) Long-term suppression in rheumatoid arthritis
d) Topical use in eczema
e) Emergency management of anaphylaxis

Answer

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is sufficient to inhibit corticotropin secretion for 24 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

Notes

In comparing the relative potencies of corticosteroids in terms of their anti-inflammatory effects, it is worth noting that high glucocorticoid activity in itself is of no advantage unless it is accompanied by relatively low mineralocorticoid activity.

Anti-inflammatory potency

Equivalent anti-inflammatory doses of corticosteroids:

Prednisolone 5 mg = Dexamethasone 750 micrograms = Hydrocortisone 20 mg

This does not take into account their mineralocorticoid effects, nor their duration of action.

Side effects

Overdosage or prolonged use can exaggerate some of the normal physiological actions of corticosteroids leading to mineralocorticoid and glucocorticoid side effects. Mineralocorticoid side effects are most marked with fludrocortisone, but are significant with hydrocortisone, negligible with the high potency glucocorticoids, betamethasone and dexamethasone, and occur only slightly with methylprednisolone and prednisolone.

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Side effects can be minimised by using the lowest effective dose for the minimum period possible. The suppressive action of a corticosteroid on cortisol secretion is least when it is given as a single dose in the morning. Whenever possible local treatment with creams, intra-articular injections, inhalations, eye drops, or enemas should be used in preference to systemic treatment.

Adrenal suppression

During prolonged therapy with corticosteroids, particularly with systemic use, adrenal atrophy develops and can persist for years after stopping. Abrupt withdrawal after a prolonged period can lead to acute adrenal insufficiency, hypotension, or death. Patients removed from long-term glucocorticoid therapy must be weaned off the drug over several days, using progressively lower doses to allow recovery of adrenal responsiveness.

To compensate for a diminished adrenocortical response caused by prolonged corticosteroid treatment, any significant intercurrent illness, trauma, or surgical procedure requires a temporary increase in corticosteroid dose, or if already stopped, a temporary reintroduction of corticosteroid treatment.

Patients on long-term corticosteroid treatment should carry a steroid treatment card which gives guidance on minimising risk and provides details of prescriber, drug, dosage and duration of treatment.

Infections

Prolonged courses of corticosteroids increase susceptibility to infections and severity of infections; clinical presentation of infections may also be atypical.

Unless they have had chickenpox, patients receiving oral or parenteral corticosteroids for purposes other than replacement should be regarded as being at risk of severe chickenpox. Manifestations of fulminant illness include pneumonia, hepatitis and disseminated intravascular coagulation; rash is not necessarily a prominent feature. Passive immunisation with varicella–zoster immunoglobulin is needed for exposed non–immune patients receiving systemic corticosteroids or for those who have used them within the previous 3 months. Confirmed chickenpox warrants specialist care and urgent treatment. Corticosteroids should not be stopped and dosage may need to be increased.

Patients taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Hydrocortisone

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Prednisolone

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Dexamethasone

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is sufficient to inhibit corticotropin secretion for 24 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

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Pharmacology: Respiratory and Endocrine

Question 20 of 76

What is the mechanism of action of salbutamol:

a Muscarinic antagonist

b Selective beta-1 agonist

c Selective beta-2 agonist

d Alpha-1 antagonist

e Alpha-2 agonist

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Pharmacology: Respiratory and Endocrine



Question 20 of 76

What is the mechanism of action of salbutamol:

a) Muscarinic antagonist
b) Selective beta-1 agonist
c) **Selective beta-2 agonist** ✔
d) Alpha-1 antagonist
e) Alpha-2 agonist

Answer

Salbutamol is a selective beta-2 agonist which acts directly on beta-2 receptors causing smooth muscle relaxation and dilation of the airways.

Notes

Selective beta-2 agonists act directly on beta-2 receptors, causing smooth muscle relaxation and dilation of the airways.

Mild to moderate symptoms of asthma respond rapidly to the inhalation of a selective short-acting beta2 agonist such as salbutamol or terbutaline sulfate. Short-acting beta-2 agonists have a rapid onset of action (15 minutes) and their effects last for up to 4 hours. Salbutamol or terbutaline sulfate can be given intravenously for severe or life-threatening acute asthma; patients should be carefully monitored and the dose adjusted according to response and heart rate.

Short acting beta-2 agonists are used for immediate relief of asthma symptoms, while some long-acting β2 agonists (e.g. salmeterol) are added to an inhaled corticosteroid in patients requiring prophylactic treatment.

Cautions

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Interactions

Hypokalaemia may be potentiated by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics. This in turn may predispose to toxicity in patients taking digoxin.

Side effects

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
- Hypokalaemia
- Cardiac arrhythmia and paradoxical bronchospasm (rare)
- Acute angle-closure glaucoma
- QT-interval prolongation

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Pharmacology: Respiratory and Endocrine

Question 21 of 76

Regarding the management of diabetic ketoacidosis (DKA), which of the following statements is INCORRECT:

a Potassium should be included in the fluid as long as the potassium level remains < 5.5 mmol/L.

b Established subcutaneous long-acting insulin therapy should be continued concomitantly.

c Blood ketone concentration should fall by at least 0.5 mmol/litre/hour.

d Blood glucose concentration should fall by at least 3 mmol/litre/hour.

e Insulin may be discontinued once blood glucose has fallen below 10 mmol/L.

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Pharmacology: Respiratory and Endocrine

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c) Blood ketone concentration should fall by at least 0.5 mmol/litre/hour.

d) Blood glucose concentration should fall by at least 3 mmol/litre/hour.

e) Insulin may be discontinued once blood glucose has fallen below 10 mmol/L. ✔

Answer

The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

Notes

Diabetic ketoacidosis (DKA) consists of the biochemical triad of ketonaemia (ketosis), hyperglycaemia, and acidaemia.

Pathophysiology

DKA usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones (i.e. glucagon, cortisol, growth hormone, catecholamines). This type of hormonal imbalance enhances hepatic gluconeogenesis and glycogenolysis resulting in severe hyperglycaemia. Enhanced lipolysis increases serum free fatty acids that are then metabolised as an alternative energy source in the process of ketogenesis. This results in accumulation of large quantities of ketone bodies and subsequent metabolic acidosis. Fluid depletion occurs due to osmotic diuresis secondary to hyperglycaemia, vomiting, and inability to take in fluid due to a diminished level of consciousness.

Diagnosis

- Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
- Blood glucose > 11.0mmol/L or known diabetes mellitus
- Bicarbonate (HCO3-) < 15.0mmol/L and/or venous pH < 7.3

Management of diabetic ketoacidosis in adults

- Intravenous fluids
  - If SBP is below 90 mmHg (adjusted for age, sex, and medication as appropriate), 500 mL sodium chloride 0.9% should be given by intravenous infusion over 10 – 15 minutes, and repeated if SBP still < 90 mmHg
  - When SBP is greater than 90 mmHg, sodium chloride infusion should be continued at a rate that replaces deficit and provides maintenance.
- Potassium replacement
  - Potassium chloride (40 mmol/L) should be included in the fluids (as long as the serum potassium level < 5.5 mmol/L and the patient is passing urine), and the plasma potassium concentration maintained between 3.5 – 5.5 mmol/L (measured at 60 minutes, 2 hours, and 2 hourly thereafter; and hourly if outside the normal range).
- Insulin
  - An intravenous insulin infusion should be started at a concentration of 1 unit/mL, at a fixed rate of 0.1 units/kg/hour.
  - Established subcutaneous long-acting insulin therapy should be continued concomitantly.
  - Blood ketone and blood glucose concentrations should be checked hourly and the insulin infusion rate adjusted accordingly. Blood ketone concentration should fall by at least 0.5 mmol/litre/hour and blood glucose concentration should fall by at least 3 mmol/litre/hour.
- Glucose
  - Once blood-glucose concentration falls below 14 mmol/litre, glucose 10% should be given by intravenous infusion (into a large vein through a large-gauge needle) at a rate of 125 mL/hour, in addition to the sodium chloride 0.9% infusion.

The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

The management of hyperosmolar hyperglycaemic state or hyperosmolar hyperglycaemic nonketotic coma is similar to that of diabetic ketoacidosis, although lower rates of insulin infusion are usually necessary and slower rehydration may be required.

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Pharmacology: Respiratory and Endocrine

Question 22 of 76

Which of the following drugs does NOT increase plasma-theophylline levels:

a Ciprofloxacin

b Clarithromycin

c Verapamil

d Cimetidine

e Rifampicin

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Question 22 of 76

Which of the following drugs does NOT increase plasma-theophylline levels:

a) Ciprofloxacin
b) Clarithromycin
c) **Verapamil** ✖
d) Cimetidine
e) Rifampicin ✔

Answer

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

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Glucagon is contraindicated in which of the following:

a Arrhythmias

b Pheochromocytoma

c Liver disease

d Cushing's disease

e Severe hypertension

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Pharmacology: Respiratory and Endocrine



Question 23 of 76

Glucagon is contraindicated in which of the following:

a) Arrhythmias ✗
b) Pheochromocytoma ✓
c) Liver disease
d) Cushing's disease
e) Severe hypertension

Answer

Glucagon is contraindicated in pheochromocytoma.

Notes

Management of hypoglycaemia in adults

- In adults who are conscious, cooperative and can swallow:
  - Give 15 – 20 g quick acting carbohydrate of the patient's choice where possible e.g. 90 – 120 mL of Lucozade or 5 – 7 Dextrosol tablets
  - Repeat capillary blood glucose 10 – 15 minutes later
  - If blood glucose is still < 4.0 mmol/L, repeat step 1 (no more than 3 treatments in total)
  - If blood glucose remains < 4.0 mmol/L after 45 minutes or 3 cycles, consider:
    - 1 mg glucagon IM
    - IV 10% glucose infusion at 100ml/hr
- In adults who are conscious but uncooperative:
  - Give either 1.5 – 2 tubes Glucogel/Dextrogel (may repeat up to 3 times)
  - If this is ineffective give glucagon 1 mg IM (may only give once)
  - If blood glucose level remains less than 4.0 mmol/L after 45 minutes (or 3 cycles), consider IV 10% glucose infusion at 100ml/hr
- In adults who are unconscious:
  - Give either:
    - 1 mg glucagon intramuscularly (if not effective after 10 – 15 minutes, IV glucose should be given)
    - 75 – 80 ml of 20% glucose intravenously over 10 – 15 minutes
    - 150 – 160 ml of 10% glucose intravenously over 10 – 15 minutes

Once blood glucose is > 4.0 mmol/L and the patient recovered, give a long acting carbohydrate of the patient's choice where possible e.g. two biscuits, one slice of bread. Note that patients given glucagon require a larger portion of long acting carbohydrate to replenish glycogen stores (double the suggested amount).

If the hypoglycaemia was due to sulfonylurea or long acting insulin therapy then be aware that the risk of hypoglycaemia may persist for up to 24 – 36 hours following the last dose, especially if there is concurrent renal impairment.

Glucagon

Glucagon, a polypeptide hormone produced by the alpha cells of the islets of Langerhans, increases plasma-glucose by mobilising glycogen stored in the liver. Glycogen promotes glycogenolysis and gluconeogenesis.

Glucagon may take up to 15 minutes to have an effect and will be less effective in alcoholics, prolonged starvation and severe liver disease when glycogen stores are depleted. In this situation or if prolonged treatment is required, IV glucose is better. Glucagon may also be less effective in patients prescribed sulfonylurea therapy.

Glucagon may also be used as an antidote in beta-blocker overdose and in anaphylaxis in patients on beta-blockers that fail to respond to adrenaline.

Glucagon is contraindicated in pheochromocytoma.

Adverse effects include:

- Nausea and vomiting
- Hyperglycaemia
- Hypokalaemia
- Hypotension

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Pharmacology: Respiratory and Endocrine

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Which of the following is an action of glucagon:

a Inhibits gluconeogenesis

b Stimulates glycogenolysis

c Increases cellular glucose uptake

d Promotes lipogenesis

e Increases protein synthesis

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Which of the following is an action of glucagon:

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b) Stimulates glycogenolysis ✓
c) Increases cellular glucose uptake
d) Promotes lipogenesis
e) Increases protein synthesis

Answer

Glucagon, a polypeptide hormone produced by the alpha cells of the islets of Langerhans, increases plasma-glucose by mobilising glycogen stored in the liver. Glycogen promotes glycogenolysis and gluconeogenesis.

Notes

Management of hypoglycaemia in adults

- In adults who are conscious, cooperative and can swallow:
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  - If blood glucose is still < 4.0 mmol/L, repeat step 1 (no more than 3 treatments in total)
  - If blood glucose remains < 4.0 mmol/L after 45 minutes or 3 cycles, consider:
    - 1 mg glucagon IM
    - IV 10% glucose infusion at 100ml/hr
- In adults who are conscious but uncooperative:
  - Give either 1.5 – 2 tubes Glucogel/Dextrogel (may repeat up to 3 times)
  - If this is ineffective give glucagon 1 mg IM (may only give once)
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    - 150 – 160 ml of 10% glucose intravenously over 10 – 15 minutes

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If the hypoglycaemia was due to sulfonylurea or long acting insulin therapy then be aware that the risk of hypoglycaemia may persist for up to 24 – 36 hours following the last dose, especially if there is concurrent renal impairment.

Glucagon

Glucagon, a polypeptide hormone produced by the alpha cells of the islets of Langerhans, increases plasma-glucose by mobilising glycogen stored in the liver. Glycogen promotes glycogenolysis and gluconeogenesis.

Glucagon may take up to 15 minutes to have an effect and will be less effective in alcoholics, prolonged starvation and severe liver disease when glycogen stores are depleted. In this situation or if prolonged treatment is required, IV glucose is better. Glucagon may also be less effective in patients prescribed sulfonylurea therapy.

Glucagon may also be used as an antidote in beta-blocker overdose and in anaphylaxis in patients on beta-blockers that fail to respond to adrenaline.

Glucagon is contraindicated in pheochromocytoma.

Adverse effects include:

- Nausea and vomiting
- Hyperglycaemia
- Hypokalaemia
- Hypotension

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Pharmacology: Respiratory and Endocrine

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What is the initial treatment for hypoglycaemia in a conscious patient:

a 1 mg subcutaneous glucagon

b 1 mg intramuscular glucagon

c Intravenous 10% glucose infusion at rate of 100 ml/hr

d A snack e.g. biscuits or toast

e 15 – 20 g glucose orally

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What is the initial treatment for hypoglycaemia in a conscious patient:

a) **1 mg subcutaneous glucagon** ✖
b) 1 mg intramuscular glucagon
c) Intravenous 10% glucose infusion at rate of 100 ml/hr
d) A snack e.g. biscuits or toast
e) 15 – 20 g glucose orally ✔

Answer

In adults who are conscious, cooperative and can swallow:

- Give 15 – 20 g quick acting carbohydrate of the patient's choice where possible e.g. 90 – 120 mL of Lucozade or 5 – 7 Dextrosol tablets
- Repeat capillary blood glucose 10 – 15 minutes later
- If blood glucose is still < 4.0 mmol/L, repeat step 1 (no more than 3 treatments in total)
- If blood glucose remains < 4.0 mmol/L after 45 minutes or 3 cycles, consider:
  - 1 mg glucagon IM
  - IV 10% glucose infusion at 100ml/hr

Notes

Management of hypoglycaemia in adults

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  - Give 15 – 20 g quick acting carbohydrate of the patient's choice where possible e.g. 90 – 120 mL of Lucozade or 5 – 7 Dextrosol tablets
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    - 75 – 80 ml of 20% glucose intravenously over 10 – 15 minutes
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Once blood glucose is > 4.0 mmol/L and the patient recovered, give a long acting carbohydrate of the patient's choice where possible e.g. two biscuits, one slice of bread. Note that patients given glucagon require a larger portion of long acting carbohydrate to replenish glycogen stores (double the suggested amount).

If the hypoglycaemia was due to sulfonylurea or long acting insulin therapy then be aware that the risk of hypoglycaemia may persist for up to 24 – 36 hours following the last dose, especially if there is concurrent renal impairment.

Glucagon

Glucagon, a polypeptide hormone produced by the alpha cells of the islets of Langerhans, increases plasma-glucose by mobilising glycogen stored in the liver. Glycogen promotes glycogenolysis and gluconeogenesis.

Glucagon may take up to 15 minutes to have an effect and will be less effective in alcoholics, prolonged starvation and severe liver disease when glycogen stores are depleted. In this situation or if prolonged treatment is required, IV glucose is better. Glucagon may also be less effective in patients prescribed sulfonylurea therapy.

Glucagon may also be used as an antidote in beta-blocker overdose and in anaphylaxis in patients on beta-blockers that fail to respond to adrenaline.

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Adverse effects include:

- Nausea and vomiting
- Hyperglycaemia
- Hypokalaemia
- Hypotension

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Pharmacology: Respiratory and Endocrine

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What is the dose of glucagon (subcutaneously or intramuscularly) used in hypoglycaemia:

a 0.5 mg

b 0.5 microgram

c 5 mg

d 1 mg

e 10 mg

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What is the dose of glucagon (subcutaneously or intramuscularly) used in hypoglycaemia:

a) 0.5 mg ✗
b) 0.5 microgram
c) 5 mg
d) 1 mg ✓
e) 10 mg

Hypoglycaemia is a medical emergency.

Initially 10 – 20 g of glucose should be given by mouth, either in liquid form or as sugar granules/lumps (e.g. Lucozade, Coca-Cola) or as a proprietary product (e.g. GlucoGel). If necessary this may be repeated after 10 – 15 minutes.

After initial treatment, a snack (e.g. biscuits, toast) providing sustained availability of carbohydrate should be given.

If oral intake is not possible, glucagon may be given by subcutaneous or intramuscular injection (1 mg in adults). If not effective within 10 minutes, intravenous glucose should be given. Intravenous 20 % glucose infusion may be given into a large vein through a large-gauge needle; 10 % glucose infusion may be used but larger volumes are required.

Glucagon may also be used as an antidote in beta-blocker overdose, and is contraindicated in pheochromocytoma. Side effects may include:

- hypersensitivity reactions
- abdominal pain
- diarrhoea
- hypokalaemia
- hypotension
- nausea
- vomiting

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Pharmacology: Respiratory and Endocrine

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Selective beta-2 agonists predominantly have which of the following clinical effects:

a Bronchodilation

b Positive chronotropic effect

c Vasoconstriction

d Vasodilation

e Positive inotropic effect

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Selective beta-2 agonists predominantly have which of the following clinical effects:

a) **Bronchodilation** ✔
b) Positive chronotropic effect
c) Vasoconstriction
d) Vasodilation
e) Positive inotropic effect

Answer

Selective beta-2 agonists act directly on beta-2 receptors, causing smooth muscle relaxation and dilation of the airways.

Notes

Selective beta-2 agonists act directly on beta-2 receptors, causing smooth muscle relaxation and dilation of the airways.

Mild to moderate symptoms of asthma respond rapidly to the inhalation of a selective short-acting beta2 agonist such as salbutamol or terbutaline sulfate. Short-acting beta-2 agonists have a rapid onset of action (15 minutes) and their effects last for up to 4 hours. Salbutamol or terbutaline sulfate can be given intravenously for severe or life-threatening acute asthma; patients should be carefully monitored and the dose adjusted according to response and heart rate.

Short acting beta-2 agonists are used for immediate relief of asthma symptoms, while some long-acting β2 agonists (e.g. salmeterol) are added to an inhaled corticosteroid in patients requiring prophylactic treatment.

Cautions

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Interactions

Hypokalaemia may be potentiated by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics. This in turn may predispose to toxicity in patients taking digoxin.

Side effects

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
- Hypokalaemia
- Cardiac arrhythmia and paradoxical bronchospasm (rare)
- Acute angle-closure glaucoma
- QT-interval prolongation

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Pharmacology: Respiratory and Endocrine

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Which of the following reactions best describes an anaphylactic reaction:

a Type I hypersensitivity reaction

b Type II hypersensitivity reaction

c Type III hypersensitivity reaction

d Type IV hypersensitivity reaction

e Complement mediated reaction

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Pharmacology: Respiratory and Endocrine

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Which of the following reactions best describes an anaphylactic reaction:

a) Type I hypersensitivity reaction ✔
b) Type II hypersensitivity reaction
c) Type III hypersensitivity reaction
d) Type IV hypersensitivity reaction
e) Complement mediated reaction

Answer

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction.

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

Question 29 of 76

Which of the following is NOT an effect of adrenaline:

a Increased heart rate

b Increased contractility

c Vasodilation

d Vasoconstriction

e Miosis

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Pharmacology: Respiratory and Endocrine

Question 29 of 76

Which of the following is NOT an effect of adrenaline:

a) Increased heart rate
b) Increased contractility
c) **Vasodilation** ✘
d) Vasoconstriction
e) Miosis ✔

Answer

Adrenaline is a catecholamine that acts on both alpha and beta receptors and increases both heart rate and contractility (beta1 effects); it can cause peripheral vasodilation (a beta2 effect) or vasoconstriction (an alpha effect). Adrenaline causes mydriasis and reduces pressure in open-angle glaucoma.

Notes

Adrenaline is a catecholamine that acts on both alpha and beta receptors and increases both heart rate and contractility (beta1 effects); it can cause peripheral vasodilation (a beta2 effect) or vasoconstriction (an alpha effect).

Adrenaline is used in cardiopulmonary resuscitation, in the emergency management of acute allergic and anaphylactic reactions and in the management of severe croup (as a nebulised solution).

Mechanism of action in anaphylaxis

As an alpha-receptor agonist, adrenaline reverses peripheral vasodilation and increased vascular permeability reducing hypotension and oedema.

As a beta-receptor agonist it dilates bronchial airways, increases the force and rate of myocardial contraction, and suppresses histamine and leukotriene release.

Adrenaline also alleviates pruritus, urticaria, and angioedema and may relieve gastrointestinal and genitourinary symptoms associated with anaphylaxis because of its relaxer effects on the smooth muscle of the stomach, intestine, uterus and urinary bladder.

Adrenaline increases glycogenolysis, reduces glucose uptake by tissues, and inhibits insulin release in the pancreas, resulting in hyperglycemia and increased blood lactic acid.

Interactions

Severe anaphylaxis in patients taking beta-blockers may not respond to adrenaline—consider bronchodilator therapy. Furthermore, adrenaline can cause severe hypertension and bradycardia in those taking non-cardioselective beta-blockers.

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Pharmacology: Respiratory and Endocrine

Question 30 of 76

Regarding inhaled corticosteroids, which of the following statements is INCORRECT:

a Corticosteroids reduce airway inflammation, oedema and mucus secretion.

b Regular use of inhaled corticosteroids reduces the frequency of exacerbations in asthma.

c Lower doses of inhaled corticosteroids may be required in smokers.

d Symptom improvement usually occurs 3 – 7 days after initiation of treatment.

e Side effects include paradoxical bronchospasm.

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Pharmacology: Respiratory and Endocrine

Question 30 of 76

Regarding inhaled corticosteroids, which of the following statements is INCORRECT:

a) **Corticosteroids reduce airway inflammation, oedema and mucus secretion.** ✗
b) Regular use of inhaled corticosteroids reduces the frequency of exacerbations in asthma.
c) Lower doses of inhaled corticosteroids may be required in smokers. ✓
d) Symptom improvement usually occurs 3 – 7 days after initiation of treatment.
e) Side effects include paradoxical bronchospasm.

Corticosteroids reduce airway inflammation, oedema and mucus secretion. Regular use of inhaled corticosteroids (e.g. beclomethasone) reduces the risk of exacerbation of asthma. **Current and previous smoking reduces the effectiveness of inhaled corticosteroids and higher doses may be necessary.** Corticosteroid inhalers must be used regularly for maximum benefit; alleviation of symptoms usually occurs 3 to 7 days after initiation.

Side effects of inhaled corticosteroids may include:

- paradoxical bronchospasm
- candidiasis of the mouth or throat (risk reduced by rinsing mouth after using inhaler and by using a spacer device)
- increased susceptibility to infection

Systemic side effects, such as adrenal suppression, are rare but may occur at high doses.

Systemic corticosteroid therapy may be necessary during episodes of stress, such as severe infection, or if the asthma is worsening, when higher doses are needed and access of inhaled drug to small airways may be reduced.

An acute attack of asthma should be treated with a short course of an oral corticosteroid (e.g. prednisolone) or intravenous corticosteroid (e.g. hydrocortisone) if oral intake is not possible, starting with a high dose. An oral corticosteroid should normally be taken as a single dose in the morning to reduce the disturbance to circadian cortisol secretion. Dosage should always be titrated to the lowest dose that controls symptoms. Regular peak-flow measurements help to optimise the dose.

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Pharmacology: Respiratory and Endocrine

Question 31 of 76

In most acutely unwell patients, who are not at risk of hypercapnic respiratory failure, oxygen saturations should be maintained at:

a 100%

b 98 – 100%

c 94 – 98%

d 92 – 96%

e 97 – 99%

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Pharmacology: Respiratory and Endocrine



Question 31 of 76

In most acutely unwell patients, who are not at risk of hypercapnic respiratory failure, oxygen saturations should be maintained at:

a) 100%
b) 98 – 100%
c) 94 – 98% ✔
d) 92 – 96% ✖
e) 97 – 99%

Answer

In most acutely ill patients with a normal or low arterial carbon dioxide (PaCO2), oxygen saturation should be 94 – 98% oxygen saturation. However, in some clinical situations such as cardiac arrest and carbon monoxide poisoning it is more appropriate to aim for the highest possible oxygen saturation until the patient is stable.

Notes

Oxygen should be regarded as a drug. It is prescribed for hypoxaemic patients to increase alveolar oxygen tension and decrease the work of breathing. The concentration of oxygen required depends on the condition being treated; the administration of an inappropriate concentration of oxygen can have serious or even fatal consequences.

High concentration oxygen therapy

In most acutely ill patients with a normal or low arterial carbon dioxide ($PaCO_2$), oxygen saturation should be 94 – 98% oxygen saturation. However, in some clinical situations such as cardiac arrest and carbon monoxide poisoning it is more appropriate to aim for the highest possible oxygen saturation until the patient is stable.

Low concentration oxygen therapy

A lower target of 88 – 92% oxygen saturation is indicated for patients at risk of hypercapnic respiratory failure e.g. patients with COPD. Until blood gases can be measured, initial oxygen should be given using a controlled concentration of 28% or less, titrated towards the SpO2 of 88 – 92%. The aim is to provide the patient with enough oxygen to achieve an acceptable arterial oxygen tension without worsening carbon dioxide retention and respiratory acidosis.

Long term oxygen therapy

Long-term administration of oxygen (usually at least 15 hours daily) prolongs survival in some patients with COPD. Assessment for long-term oxygen therapy requires measurement of arterial blood gas tensions. A nasal cannula is usually preferred for long-term oxygen therapy from an oxygen concentrator. It can, however, produce dermatitis and mucosal drying in sensitive individuals.

Giving oxygen by nasal cannula allows the patient to talk, eat, and drink, but the concentration of oxygen is not controlled; this may not be appropriate for acute respiratory failure. Increased respiratory depression is seldom a problem in patients with stable respiratory failure treated with low concentrations of oxygen although it may occur during exacerbations; patients and relatives should be warned to call for medical help if drowsiness or confusion occur.

Intermittent oxygen therapy

Oxygen is occasionally prescribed for short-burst (intermittent) use for episodes of breathlessness not relieved by other treatment in patients with severe chronic obstructive pulmonary disease, interstitial lung disease, heart failure, and in palliative care.

Ambulatory oxygen

Ambulatory oxygen is prescribed for patients on long-term oxygen therapy who need to be away from home on a regular basis. Patients who are not on long-term oxygen therapy can be considered for ambulatory oxygen therapy if there is evidence of exercise-induced oxygen desaturation and of improvement in blood oxygen saturation and exercise capacity with oxygen.

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Pharmacology: Respiratory and Endocrine

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Which of the following presentations is NOT consistent with the diagnosis of anaphylaxis:

a Hypotension alone

b Generalised urticaria and angioedema alone

c Shock with collapse and angioedema

d Stridor, hoarseness and hypotension with no skin or mucosal changes

e Dyspnoea, tachypnoea, wheeze and hypoxia with no skin or mucosal changes

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Pharmacology: Respiratory and Endocrine

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After initial emergency treatment, which of the following tests may be used to confirm a suspected diagnosis of anaphylaxis:

a Immunoglobulins

b Mast cell tryptase

c ANCA autoantibodies

d Specific IgE (RAST) test

e Complement function assays

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Pharmacology: Respiratory and Endocrine

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After initial emergency treatment, which of the following tests may be used to confirm a suspected diagnosis of anaphylaxis:

a) **Immunoglobulins** ✗
b) Mast cell tryptase ✓
c) ANCA autoantibodies
d) Specific IgE (RAST) test
e) Complement function assays

Answer

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis. Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
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Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

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Regarding the management of diabetic ketoacidosis (DKA), insulin infusion may be stopped after which of the following:

a Blood ketone concentration is below 0.3 mmol/L

b Blood pH is above 7.3

c Patient is conscious

d Patient is able to eat and drink

e All of the above have been achieved

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Pharmacology: Respiratory and Endocrine



Question 34 of 76

Regarding the management of diabetic ketoacidosis (DKA), insulin infusion may be stopped after which of the following:

a) **Blood ketone concentration is below 0.3 mmol/L** ✗
b) Blood pH is above 7.3
c) Patient is conscious
d) Patient is able to eat and drink
e) All of the above have been achieved ✓

Answer

The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

Notes

Diabetic ketoacidosis (DKA) consists of the biochemical triad of ketonaemia (ketosis), hyperglycaemia, and acidaemia.

Pathophysiology

DKA usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones (i.e. glucagon, cortisol, growth hormone, catecholamines). This type of hormonal imbalance enhances hepatic gluconeogenesis and glycogenolysis resulting in severe hyperglycaemia. Enhanced lipolysis increases serum free fatty acids that are then metabolised as an alternative energy source in the process of ketogenesis. This results in accumulation of large quantities of ketone bodies and subsequent metabolic acidosis. Fluid depletion occurs due to osmotic diuresis secondary to hyperglycaemia, vomiting, and inability to take in fluid due to a diminished level of consciousness.

Diagnosis

- Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
- Blood glucose > 11.0mmol/L or known diabetes mellitus
- Bicarbonate (HCO_3^-) < 15.0mmol/L and/or venous pH < 7.3

Management of diabetic ketoacidosis in adults

- Intravenous fluids
  - If SBP is below 90 mmHg (adjusted for age, sex, and medication as appropriate), 500 mL sodium chloride 0.9% should be given by intravenous infusion over 10 – 15 minutes, and repeated if SBP still < 90 mmHg
  - When SBP is greater than 90 mmHg, sodium chloride infusion should be continued at a rate that replaces deficit and provides maintenance.
- Potassium replacement
  - Potassium chloride (40 mmol/L) should be included in the fluids (as long as the serum potassium level < 5.5 mmol/L and the patient is passing urine), and the plasma potassium concentration maintained between 3.5 – 5.5 mmol/L (measured at 60 minutes, 2 hours, and 2 hourly thereafter; and hourly if outside the normal range).
- Insulin
  - An intravenous insulin infusion should be started at a concentration of 1 unit/mL, at a fixed rate of 0.1 units/kg/hour.
  - Established subcutaneous long-acting insulin therapy should be continued concomitantly.
  - Blood ketone and blood glucose concentrations should be checked hourly and the insulin infusion rate adjusted accordingly. Blood ketone concentration should fall by at least 0.5 mmol/litre/hour and blood glucose concentration should fall by at least 3 mmol/litre/hour.
- Glucose
  - Once blood-glucose concentration falls below 14 mmol/litre, glucose 10% should be given by intravenous infusion (into a large vein through a large-gauge needle) at a rate of 125 mL/hour, in addition to the sodium chloride 0.9% infusion.

The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

The management of hyperosmolar hyperglycaemic state or hyperosmolar hyperglycaemic nonketotic coma is similar to that of diabetic ketoacidosis, although lower rates of insulin infusion are usually necessary and slower rehydration may be required.

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Regarding the management of diabetic ketoacidosis (DKA), insulin should initially be given:

a At a concentration of 1 unit/mL at a fixed rate of 1 units/kg/hour

b At a concentration of 0.1 units/mL at a fixed rate 0.1 units/kg/hour

c At a concentration of 1 unit/mL at a fixed rate of 0.2 units/kg/hour

d At a concentration of 0.1 units/mL at a fixed rate of 1 units/kg/hour

e At a concentration of 1 unit/mL at a fixed rate of 0.1 units/kg/hour

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Pharmacology: Respiratory and Endocrine



Question 35 of 76

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a) At a concentration of 1 unit/mL at a fixed rate of 1 units/kg/hour ✗

b) At a concentration of 0.1 units/mL at a fixed rate 0.1 units/kg/hour

c) At a concentration of 1 unit/mL at a fixed rate of 0.2 units/kg/hour

d) At a concentration of 0.1 units/mL at a fixed rate of 1 units/kg/hour

e) At a concentration of 1 unit/mL at a fixed rate of 0.1 units/kg/hour ✓

Answer

An intravenous insulin infusion should be started at a concentration of 1 unit/mL, at a fixed rate of 0.1 units/kg/hour. Established subcutaneous long-acting insulin therapy should be continued concomitantly. Blood ketone and blood glucose concentrations should be checked hourly and the insulin infusion rate adjusted accordingly. Blood ketone concentration should fall by at least 0.5 mmol/litre/hour and blood glucose concentration should fall by at least 3 mmol/litre/hour.

Notes

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The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

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Pharmacology: Respiratory and Endocrine

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Theophylline is used for which of the following clinical effects:

a Vasodilation

b Vasoconstriction

c Bronchodilation

d Anti-inflammatory

e Anti-motility

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Pharmacology: Respiratory and Endocrine



Question 36 of 76

Theophylline is used for which of the following clinical effects:

a) Vasodilation
b) Vasoconstriction
c) **Bronchodilation** ✔
d) Anti-inflammatory
e) Anti-motility

Answer

Theophylline is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

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Which of the following clinical features would characterise a severe asthma attack in an adult as life-threatening:

a Use of accessory respiratory muscles

b PEF 35% of best/predicted

c PaO 8.5 kPa

d PaCO 5.0 kPa

e HR 120 bpm

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a) **Use of accessory respiratory muscles** ✗
b) PEF 35% of best/predicted
c) PaO 8.5 kPa
d) PaCO 5.0 kPa ✓
e) HR 120 bpm

Answer

Life-threatening acute asthma is defined as any one of the following in someone with severe asthma:

- PEFR < 33% of best or predicted
- SpO2< 92% or PaO2 < 8 kPa
- Normal PaCO2 (4.6 – 6.0 kPa)
- Silent chest
- Cyanosis
- Poor respiratory effort
- Arrhythmia
- Hypotension
- Exhaustion
- Altered conscious level

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack.
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

Question 38 of 76

A 31 year old patient with a known nut allergy was eating at a Thai restaurant when he suddenly complained of lip and tongue swelling and difficulty breathing. He is brought to ED by ambulance with suspected anaphylaxis. He has received intramuscular adrenaline in the ambulance. What are the most appropriate doses of the second line treatments for anaphylaxis:

a 5 mg chlorphenamine and 200 mg hydrocortisone

b 10 mg chlorphenamine and 200 mg hydrocortisone

c 10 mg chlorphenamine and 300 mg hydrocortisone

d 20 mg chlorphenamine and 400 mg hydrocortisone

e 20 chlorphenamine and 300 mg hydrocortisone

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a) 5 mg chlorphenamine and 200 mg hydrocortisone ✖
b) 10 mg chlorphenamine and 200 mg hydrocortisone ✔
c) 10 mg chlorphenamine and 300 mg hydrocortisone
d) 20 mg chlorphenamine and 400 mg hydrocortisone
e) 20 chlorphenamine and 300 mg hydrocortisone

Answer

Intravenous or intramuscular chlorphenamine and hydrocortisone should be given as second line drugs to reduce the severity and duration of symptoms, at a dose of 10 mg chlorphenamine and 200 mg hydrocortisone in adults.

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

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Which of the following is NOT a feature of theophylline toxicity:

a Severe intractable vomiting

b Agitation

c Dilated pupils

d Sinus tachycardia

e Hyperkalaemia

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Pharmacology: Respiratory and Endocrine



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Which of the following is NOT a feature of theophylline toxicity:

a) Severe intractable vomiting
b) Agitation
c) **Dilated pupils** ✗
d) Sinus tachycardia
e) Hyperkalaemia ✓

Answer

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

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Which of the following is NOT an action of insulin:

a Increased gluconeogenesis

b Increased glycogenesis

c Increased lipogenesis

d Increased cellular potassium uptake

e Increased protein synthesis

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a) **Increased gluconeogenesis** ✔
b) Increased glycogenesis
c) Increased lipogenesis
d) Increased cellular potassium uptake
e) Increased protein synthesis

Answer

Insulin acts to:

- Increase cellular glucose uptake
- Decrease gluconeogenesis
- Increase glycogenesis/decrease glycogenolysis
- Increase lipogenesis/decrease lipolysis
- Increase protein synthesis/decrease proteolysis
- Increase cellular potassium uptake

Notes

Insulin action

Insulin plays a key role in the regulation of carbohydrate, fat, and protein metabolism.

Insulin acts to:

- Increase cellular glucose uptake
- Decrease gluconeogenesis
- Increase glycogenesis/decrease glycogenolysis
- Increase lipogenesis/decrease lipolysis
- Increase protein synthesis/decrease proteolysis
- Increase cellular potassium uptake

Insulin is needed for all patients with type 1 DM and some patients with type 2 DM where other methods have failed to achieve good control, and temporarily in the presence of intercurrent illness or perioperatively.

Insulin administration

Insulin is inactivated by gastro-intestinal enzymes, and must therefore be given by injection; the subcutaneous route is ideal in most circumstances. Insulin is usually injected into the upper arms, thighs, buttocks, or abdomen; absorption from a limb site may be increased if the limb is used in strenuous exercise after the injection. Generally subcutaneous insulin injections cause few problems; lipodystrophy may occur but can be minimised by using different injection sites in rotation. Local allergic reactions are rare.

Insulin preparations

Insulin preparations can be divided into three types:

1. those of short duration which have a relatively rapid onset of action, namely soluble insulin (most appropriate form of insulin for use in DKA – given as intravenous solution) and the rapid-acting insulin analogues (insulin aspart, insulin glulisine, and insulin lispro)
2. those with an intermediate action (e.g. isophane insulin)
3. those whose action is slower in onset and lasts for long periods (e.g. protamine zinc insulin, insulin detemir, and insulin glargine)

Soluble insulin: When injected subcutaneously, soluble insulin has a rapid onset of action (30 to 60 minutes), a peak action between 2 and 4 hours, and a duration of action of up to 8 hours. When injected intravenously, soluble insulin has a very short half-life of only about 5 minutes and its effect disappears within 30 minutes.

The rapid-acting human insulin analogues have a faster onset and shorter duration of action than soluble insulin; as a result, compared to soluble insulin, fasting and preprandial blood-glucose concentrations are a little higher, postprandial blood-glucose concentration is a little lower, and hypoglycaemia occurs slightly less frequently.

When given by subcutaneous injection, intermediate- and long-acting insulins have an onset of action of approximately 1–2 hours, a maximal effect at 4–12 hours, and a duration of 16–42 hours.

Treatment regimes

The aim of treatment is to achieve the best possible control of blood-glucose concentration whilst avoiding disabling hypoglycaemia. Mixtures of insulin preparations may be required and appropriate combinations have to be determined for the individual patient.

Insulin regimes should be tailored to the requirements of the individual and may include:

- once/twice daily SC injections (often with a premixed solution of short-acting insulin or rapid-acting insulin analogue with an intermediate-acting or long-acting insulin)
- multiple daily SC injections (with a short-acting insulin or a rapid-acting insulin analogue given before meals with intermediate-acting or long-acting insulin once or twice daily)
- continuous subcutaneous insulin infusion

Insulin requirements may be increased by infection, stress, accidental or surgical trauma, and during puberty. Requirements may be decreased in those with certain endocrine disorders (e.g. Addison's disease, hypopituitarism), or in coeliac disease.

Implications of diabetes on driving

Loss of warning of hypoglycaemia among insulin-treated patients can be a serious hazard, especially for drivers and those in dangerous occupations. Very tight control of diabetes leads to increased risk of hypoglycaemic episodes and an increase in the frequency of hypoglycaemia episodes may blunt hypoglycaemic awareness. Beta-blockers can also blunt hypoglycaemic awareness. Drivers with diabetes may be required to notify the DVLA of their condition, depending on their treatment, the type of licence, and whether they have diabetic complications.

Oral antidiabetic drugs

Oral antidiabetic drugs are used for the treatment of type 2 diabetes mellitus. They should be prescribed only if the patient fails to respond adequately to at least 3 months' restriction of energy and carbohydrate intake and an increase in physical activity. They should be used to augment the effect of diet and exercise, and not to replace them.

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Pharmacology: Respiratory and Endocrine

Question 41 of 76

In acutely unwell patients who are at risk of hypercapnic respiratory failure, oxygen saturations should be maintained at:

a 88 – 90%

b 96 – 98%

c 92 – 94%

d 88 – 92%

e 94 – 98%

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Pharmacology: Respiratory and Endocrine

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In acutely unwell patients who are at risk of hypercapnic respiratory failure, oxygen saturations should be maintained at:

a) 88 – 90%
b) 96 – 98%
c) **92 – 94%** ✗
d) 88 – 92% ✓
e) 94 – 98%

Answer

A lower target of 88 – 92% oxygen saturation is indicated for patients at risk of hypercapnic respiratory failure e.g. patients with COPD. Until blood gases can be measured, initial oxygen should be given using a controlled concentration of 28% or less, titrated towards the SpO2 of 88 – 92%. The aim is to provide the patient with enough oxygen to achieve an acceptable arterial oxygen tension without worsening carbon dioxide retention and respiratory acidosis.

Notes

Oxygen should be regarded as a drug. It is prescribed for hypoxaemic patients to increase alveolar oxygen tension and decrease the work of breathing. The concentration of oxygen required depends on the condition being treated; the administration of an inappropriate concentration of oxygen can have serious or even fatal consequences.

High concentration oxygen therapy

In most acutely ill patients with a normal or low arterial carbon dioxide ($PaCO_2$), oxygen saturation should be 94 – 98% oxygen saturation. However, in some clinical situations such as cardiac arrest and carbon monoxide poisoning it is more appropriate to aim for the highest possible oxygen saturation until the patient is stable.

Low concentration oxygen therapy

A lower target of 88 – 92% oxygen saturation is indicated for patients at risk of hypercapnic respiratory failure e.g. patients with COPD. Until blood gases can be measured, initial oxygen should be given using a controlled concentration of 28% or less, titrated towards the SpO2 of 88 – 92%. The aim is to provide the patient with enough oxygen to achieve an acceptable arterial oxygen tension without worsening carbon dioxide retention and respiratory acidosis.

Long term oxygen therapy

Long-term administration of oxygen (usually at least 15 hours daily) prolongs survival in some patients with COPD. Assessment for long-term oxygen therapy requires measurement of arterial blood gas tensions. A nasal cannula is usually preferred for long-term oxygen therapy from an oxygen concentrator. It can, however, produce dermatitis and mucosal drying in sensitive individuals.

Giving oxygen by nasal cannula allows the patient to talk, eat, and drink, but the concentration of oxygen is not controlled; this may not be appropriate for acute respiratory failure. Increased respiratory depression is seldom a problem in patients with stable respiratory failure treated with low concentrations of oxygen although it may occur during exacerbations; patients and relatives should be warned to call for medical help if drowsiness or confusion occur.

Intermittent oxygen therapy

Oxygen is occasionally prescribed for short-burst (intermittent) use for episodes of breathlessness not relieved by other treatment in patients with severe chronic obstructive pulmonary disease, interstitial lung disease, heart failure, and in palliative care.

Ambulatory oxygen

Ambulatory oxygen is prescribed for patients on long-term oxygen therapy who need to be away from home on a regular basis. Patients who are not on long-term oxygen therapy can be considered for ambulatory oxygen therapy if there is evidence of exercise-induced oxygen desaturation and of improvement in blood oxygen saturation and exercise capacity with oxygen.

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Pharmacology: Respiratory and Endocrine

Question 42 of 76

What is the mechanism of action of ipratropium bromide:

a Beta-2 agonist

b Muscarinic antagonist

c Muscarinic agonist

d Beta-blocker

e Alpha-agonist

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Pharmacology: Respiratory and Endocrine

Question 42 of 76

What is the mechanism of action of ipratropium bromide:

a) Beta-2 agonist
b) **Muscarinic antagonist** ✔
c) Muscarinic agonist
d) Beta-blocker
e) Alpha-agonist

Answer

Ipratropium bromide, a short-acting antimuscarinic bronchodilator, causes bronchodilation by blocking the cholinergic nerves in the airways.

Notes

Ipratropium bromide, a short-acting antimuscarinic bronchodilator, causes bronchodilation by blocking the cholinergic nerves in the airways.

Ipratropium bromide can provide short-term relief in chronic asthma, but short-acting beta-2 agonists act more quickly and are preferred. Ipratropium bromide by nebulisation can be added to other standard treatment in life-threatening asthma or if acute asthma fails to improve with standard therapy.

The aerosol inhalation of ipratropium bromide may be used for short-term relief in mild COPD in patients who are not using a long-acting antimuscarinic drug. Its maximal effect occurs 30 – 60 minutes after use; its duration of action is 3 to 6 hours and bronchodilation can usually be maintained with treatment 3 times a day.

Cautions

Ipratropium bromide should be used with caution in:

- Men with prostatic hyperplasia and bladder-outflow obstruction (worsened urinary retention has been reported in elderly men)
- People with chronic kidney disease (CKD) stages 3 and above (because of the risk of drug toxicity)
- People with angle-closure glaucoma (nebulised mist of antimuscarinic drugs can precipitate or worsen acute angle-closure glaucoma)

Interactions

There are no important drug interactions with inhaled muscarinic antagonists.

Side effects

Inhaled antimuscarinics are generally well tolerated as they are poorly absorbed systemically.

Their adverse effects include:

- Dry mouth and abnormal taste in the mouth
- Nasal congestion and dryness of nasal mucosa
- Acute angle-closure glaucoma (reported in people on nebulised ipratropium)

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Pharmacology: Respiratory and Endocrine

Question 43 of 76

Which of the following is NOT a glucocorticoid effect of corticosteroids:

a Hyperglycaemia

b Osteoporosis

c Muscle wasting

d Peptic ulceration

e Hypertension

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Pharmacology: Respiratory and Endocrine

Question 43 of 76

Which of the following is NOT a glucocorticoid effect of corticosteroids:

a) Hyperglycaemia
b) Osteoporosis
c) Muscle wasting
d) Peptic ulceration
e) **Hypertension** ✔

Answer

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Notes

In comparing the relative potencies of corticosteroids in terms of their anti-inflammatory effects, it is worth noting that high glucocorticoid activity in itself is of no advantage unless it is accompanied by relatively low mineralocorticoid activity.

Anti-inflammatory potency

Equivalent anti-inflammatory doses of corticosteroids:

Prednisolone 5 mg = Dexamethasone 750 micrograms = Hydrocortisone 20 mg

This does not take into account their mineralocorticoid effects, nor their duration of action.

Side effects

Overdosage or prolonged use can exaggerate some of the normal physiological actions of corticosteroids leading to mineralocorticoid and glucocorticoid side effects. Mineralocorticoid side effects are most marked with fludrocortisone, but are significant with hydrocortisone, negligible with the high potency glucocorticoids, betamethasone and dexamethasone, and occur only slightly with methylprednisolone and prednisolone.

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Side effects can be minimised by using the lowest effective dose for the minimum period possible. The suppressive action of a corticosteroid on cortisol secretion is least when it is given as a single dose in the morning. Whenever possible local treatment with creams, intra-articular injections, inhalations, eye drops, or enemas should be used in preference to systemic treatment.

Adrenal suppression

During prolonged therapy with corticosteroids, particularly with systemic use, adrenal atrophy develops and can persist for years after stopping. Abrupt withdrawal after a prolonged period can lead to acute adrenal insufficiency, hypotension, or death. Patients removed from long-term glucocorticoid therapy must be weaned off the drug over several days, using progressively lower doses to allow recovery of adrenal responsiveness.

To compensate for a diminished adrenocortical response caused by prolonged corticosteroid treatment, any significant intercurrent illness, trauma, or surgical procedure requires a temporary increase in corticosteroid dose, or if already stopped, a temporary reintroduction of corticosteroid treatment.

Patients on long-term corticosteroid treatment should carry a steroid treatment card which gives guidance on minimising risk and provides details of prescriber, drug, dosage and duration of treatment.

Infections

Prolonged courses of corticosteroids increase susceptibility to infections and severity of infections; clinical presentation of infections may also be atypical.

Unless they have had chickenpox, patients receiving oral or parenteral corticosteroids for purposes other than replacement should be regarded as being at risk of severe chickenpox. Manifestations of fulminant illness include pneumonia, hepatitis and disseminated intravascular coagulation; rash is not necessarily a prominent feature. Passive immunisation with varicella–zoster immunoglobulin is needed for exposed non–immune patients receiving systemic corticosteroids or for those who have used them within the previous 3 months. Confirmed chickenpox warrants specialist care and urgent treatment. Corticosteroids should not be stopped and dosage may need to be increased.

Patients taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Hydrocortisone

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Prednisolone

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Dexamethasone

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is sufficient to inhibit corticotropin secretion for 24 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

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Pharmacology: Respiratory and Endocrine

Question 44 of 76

What is the mechanism of action of chlorphenamine:

a H1-receptor antagonist

b Histamine agonist

c H2-receptor antagonist

d Leukotriene receptor antagonist

e Muscarinic antagonist

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Pharmacology: Respiratory and Endocrine



Question 44 of 76

What is the mechanism of action of chlorphenamine:

a) **H1-receptor antagonist** ✔
b) Histamine agonist
c) H2-receptor antagonist
d) Leukotriene receptor antagonist
e) Muscarinic antagonist

Answer

Chlorphenamine is a competitive inhibitor at the H1-receptor (an anti-histamine).

Notes

Antihistamines are competitive inhibitors at the H1-receptor (in contrast to H2 receptor antagonists used to decrease gastric acid secretion). They act to relax histamine-induced bronchoconstriction, block the vasodilator effect of histamine, inhibit histamine-induced increases in capillary permeability and block mucus secretion and sensory nerve stimulation.

Histamine (H1)-receptor antagonists are well absorbed after oral administration. The effects of these agents are usually seen in 30 minutes (with maximal effects at 1 – 2 h); the duration of action is 3 – 8 hours for first-generation compounds and 3 – 24 hours for second-generation compounds.

H1-receptor antagonists are metabolised in the liver; many induce microsomal enzymes and alter their own metabolism and that of other drugs.

Indications

- Allergic rhinitis and conjunctivitis
- Urticarial rashes, pruritus, insect bites and stings
- Angioedema
- Anaphylaxis (second line adjunct to adrenaline)
- Nausea/vomiting and prevention of motion sickness
- Insomnia

Chlorphenamine

All older antihistamines cause sedation but alimemazine tartrate and promethazine may be more sedating whereas chlorphenamine maleate and cyclizine may be less so. This sedating activity is sometimes used to manage the pruritus associated with some allergies or used to manage occasional insomnia. There is little evidence that any one of the older, 'sedating' antihistamines is superior to another and patients vary widely in their response.

Cetirizine

The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent.

Cautions

Antihistamines should usually be avoided in acute porphyrias (although some antihistamines are thought to be safe).

Antihistamines should be used with caution in epilepsy, prostatic hypertrophy, urinary hypertension, hepatic impairment and susceptibility to angle-closure glaucoma.

Side effects (significantly reduced with second-generation agents)

Elderly patients and children are more susceptible to side effects.

Common side effects of antihistamines may include:

- Anticholinergic effects (blurred vision, dry mouth, urinary retention)
- Headache
- Gastrointestinal disturbances
- Psychomotor impairment (sedation, dizziness and loss of appetite)

Drowsiness may affect performance of skilled tasks (e.g. cycling or driving); sedating effects are enhanced by alcohol and opioid analgesics.

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Pharmacology: Respiratory and Endocrine

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Regarding the management of diabetic ketoacidosis (DKA), glucose should be given in addition to insulin once blood-glucose concentration falls below:

a 20 mmol/L

b 18 mmol/L

c 14 mmol/L

d 10 mmol/L

e 8 mmol/L

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Regarding the management of diabetic ketoacidosis (DKA), glucose should be given in addition to insulin once blood-glucose concentration falls below:

a) 20 mmol/L
b) **18 mmol/L** ✗
c) 14 mmol/L ✓
d) 10 mmol/L
e) 8 mmol/L

Answer

Once blood-glucose concentration falls below 14 mmol/litre, glucose 10% should be given by intravenous infusion (into a large vein through a large-gauge needle) at a rate of 125 mL/hour, in addition to the sodium chloride 0.9% infusion.

Notes

Diabetic ketoacidosis (DKA) consists of the biochemical triad of ketonaemia (ketosis), hyperglycaemia, and acidaemia.

Pathophysiology

DKA usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones (i.e. glucagon, cortisol, growth hormone, catecholamines). This type of hormonal imbalance enhances hepatic gluconeogenesis and glycogenolysis resulting in severe hyperglycaemia. Enhanced lipolysis increases serum free fatty acids that are then metabolised as an alternative energy source in the process of ketogenesis. This results in accumulation of large quantities of ketone bodies and subsequent metabolic acidosis. Fluid depletion occurs due to osmotic diuresis secondary to hyperglycaemia, vomiting, and inability to take in fluid due to a diminished level of consciousness.

Diagnosis

- Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
- Blood glucose > 11.0mmol/L or known diabetes mellitus
- Bicarbonate (HCO_3^-) < 15.0mmol/L and/or venous pH < 7.3

Management of diabetic ketoacidosis in adults

- Intravenous fluids
  - If SBP is below 90 mmHg (adjusted for age, sex, and medication as appropriate), 500 mL sodium chloride 0.9% should be given by intravenous infusion over 10 – 15 minutes, and repeated if SBP still < 90 mmHg
  - When SBP is greater than 90 mmHg, sodium chloride infusion should be continued at a rate that replaces deficit and provides maintenance.
- Potassium replacement
  - Potassium chloride (40 mmol/L) should be included in the fluids (as long as the serum potassium level < 5.5 mmol/L and the patient is passing urine), and the plasma potassium concentration maintained between 3.5 – 5.5 mmol/L (measured at 60 minutes, 2 hours, and 2 hourly thereafter; and hourly if outside the normal range).
- Insulin
  - An intravenous insulin infusion should be started at a concentration of 1 unit/mL, at a fixed rate of 0.1 units/kg/hour.
  - Established subcutaneous long-acting insulin therapy should be continued concomitantly.
  - Blood ketone and blood glucose concentrations should be checked hourly and the insulin infusion rate adjusted accordingly. Blood ketone concentration should fall by at least 0.5 mmol/litre/hour and blood glucose concentration should fall by at least 3 mmol/litre/hour.
- Glucose
  - Once blood-glucose concentration falls below 14 mmol/litre, glucose 10% should be given by intravenous infusion (into a large vein through a large-gauge needle) at a rate of 125 mL/hour, in addition to the sodium chloride 0.9% infusion.

The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

The management of hyperosmolar hyperglycaemic state or hyperosmolar hyperglycaemic nonketotic coma is similar to that of diabetic ketoacidosis, although lower rates of insulin infusion are usually necessary and slower rehydration may be required.

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Pharmacology: Respiratory and Endocrine

Question 46 of 76

Regarding selective beta-2 agonists, which of the following statements is INCORRECT:

a Hypokalaemia may be potentiated by concomitant treatment with theophylline.

b Beta-2 agonists may inhibit thyroid activity.

c Beta-2 agonists should be used with caution in diabetes due to a risk of hyperglycaemia and ketoacidosis.

d Beta-2 agonists should be used with caution in those with a susceptibility to QT-interval prolongation.

e Beta-2 agonists cause an increased risk of arrhythmias and should be used with caution in patients with cardiovascular disease.

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Pharmacology: Respiratory and Endocrine



Question 46 of 76

Regarding selective beta-2 agonists, which of the following statements is INCORRECT:

a) **Hypokalaemia may be potentiated by concomitant treatment with theophylline.** ✗

b) Beta-2 agonists may inhibit thyroid activity. ✓

c)
Beta-2 agonists should be used with caution in diabetes due to a risk of hyperglycaemia and ketoacidosis.

d)
Beta-2 agonists should be used with caution in those with a susceptibility to QT-interval prolongation.

e)
Beta-2 agonists cause an increased risk of arrhythmias and should be used with caution in patients with cardiovascular disease.

Answer

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Notes

Selective beta-2 agonists act directly on beta-2 receptors, causing smooth muscle relaxation and dilation of the airways.

Mild to moderate symptoms of asthma respond rapidly to the inhalation of a selective short-acting beta2 agonist such as salbutamol or terbutaline sulfate. Short-acting beta-2 agonists have a rapid onset of action (15 minutes) and their effects last for up to 4 hours. Salbutamol or terbutaline sulfate can be given intravenously for severe or life-threatening acute asthma; patients should be carefully monitored and the dose adjusted according to response and heart rate.

Short acting beta-2 agonists are used for immediate relief of asthma symptoms, while some long-acting β2 agonists (e.g. salmeterol) are added to an inhaled corticosteroid in patients requiring prophylactic treatment.

Cautions

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Interactions

Hypokalaemia may be potentiated by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics. This in turn may predispose to toxicity in patients taking digoxin.

Side effects

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
- Hypokalaemia
- Cardiac arrhythmia and paradoxical bronchospasm (rare)
- Acute angle-closure glaucoma
- QT-interval prolongation

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Pharmacology: Respiratory and Endocrine

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Regarding adrenaline, which of the following statements is CORRECT:

a Adrenaline only acts on alpha receptors.

b Adrenaline only acts on beta receptors.

c Adrenaline has positive chronotropic but negative inotropic effects.

d Adrenaline can cause vasodilation or vasoconstriction.

e Adrenaline is only used in the management of cardiopulmonary resuscitation.

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Pharmacology: Respiratory and Endocrine

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Regarding adrenaline, which of the following statements is CORRECT:

a) **Adrenaline only acts on alpha receptors.** ✘
b) Adrenaline only acts on beta receptors.
c) Adrenaline has positive chronotropic but negative inotropic effects.
d) Adrenaline can cause vasodilation or vasoconstriction. ✔
e) Adrenaline is only used in the management of cardiopulmonary resuscitation.

Answer

Adrenaline is a catecholamine that acts on both alpha and beta receptors and increases both heart rate and contractility (beta1 effects); it can cause peripheral vasodilation (a beta2 effect) or vasoconstriction (an alpha effect). Adrenaline is used in cardiopulmonary resuscitation, in the emergency management of acute allergic and anaphylactic reactions and in the management of severe croup (as a nebulised solution).

Notes

Adrenaline is a catecholamine that acts on both alpha and beta receptors and increases both heart rate and contractility (beta1 effects); it can cause peripheral vasodilation (a beta2 effect) or vasoconstriction (an alpha effect).

Adrenaline is used in cardiopulmonary resuscitation, in the emergency management of acute allergic and anaphylactic reactions and in the management of severe croup (as a nebulised solution).

Mechanism of action in anaphylaxis

As an alpha-receptor agonist, adrenaline reverses peripheral vasodilation and increased vascular permeability reducing hypotension and oedema.

As a beta-receptor agonist it dilates bronchial airways, increases the force and rate of myocardial contraction, and suppresses histamine and leukotriene release.

Adrenaline also alleviates pruritus, urticaria, and angioedema and may relieve gastrointestinal and genitourinary symptoms associated with anaphylaxis because of its relaxer effects on the smooth muscle of the stomach, intestine, uterus and urinary bladder.

Adrenaline increases glycogenolysis, reduces glucose uptake by tissues, and inhibits insulin release in the pancreas, resulting in hyperglycemia and increased blood lactic acid.

Interactions

Severe anaphylaxis in patients taking beta-blockers may not respond to adrenaline—consider bronchodilator therapy. Furthermore, adrenaline can cause severe hypertension and bradycardia in those taking non-cardioselective beta-blockers.

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Pharmacology: Respiratory and Endocrine

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Which of the following has the most potent glucocorticoid effect:

a Prednisolone

b Fludrocortisone

c Hydrocortisone

d Methylprednisolone

e Dexamethasone

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Pharmacology: Respiratory and Endocrine

Question 48 of 76

Which of the following has the most potent glucocorticoid effect:

a) **Prednisolone** ✗
b) Fludrocortisone
c) Hydrocortisone
d) Methylprednisolone
e) Dexamethasone ✓

Answer

Dexamethasone and betamethasone have the most potent glucocorticoid (anti-inflammatory) effect.

Notes

In comparing the relative potencies of corticosteroids in terms of their anti-inflammatory effects, it is worth noting that high glucocorticoid activity in itself is of no advantage unless it is accompanied by relatively low mineralocorticoid activity.

Anti-inflammatory potency

Equivalent anti-inflammatory doses of corticosteroids:

Prednisolone 5 mg = Dexamethasone 750 micrograms = Hydrocortisone 20 mg

This does not take into account their mineralocorticoid effects, nor their duration of action.

Side effects

Overdosage or prolonged use can exaggerate some of the normal physiological actions of corticosteroids leading to mineralocorticoid and glucocorticoid side effects. Mineralocorticoid side effects are most marked with fludrocortisone, but are significant with hydrocortisone, negligible with the high potency glucocorticoids, betamethasone and dexamethasone, and occur only slightly with methylprednisolone and prednisolone.

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Side effects can be minimised by using the lowest effective dose for the minimum period possible. The suppressive action of a corticosteroid on cortisol secretion is least when it is given as a single dose in the morning. Whenever possible local treatment with creams, intra-articular injections, inhalations, eye drops, or enemas should be used in preference to systemic treatment.

Adrenal suppression

During prolonged therapy with corticosteroids, particularly with systemic use, adrenal atrophy develops and can persist for years after stopping. Abrupt withdrawal after a prolonged period can lead to acute adrenal insufficiency, hypotension, or death. Patients removed from long-term glucocorticoid therapy must be weaned off the drug over several days, using progressively lower doses to allow recovery of adrenal responsiveness.

To compensate for a diminished adrenocortical response caused by prolonged corticosteroid treatment, any significant intercurrent illness, trauma, or surgical procedure requires a temporary increase in corticosteroid dose, or if already stopped, a temporary reintroduction of corticosteroid treatment.

Patients on long-term corticosteroid treatment should carry a steroid treatment card which gives guidance on minimising risk and provides details of prescriber, drug, dosage and duration of treatment.

Infections

Prolonged courses of corticosteroids increase susceptibility to infections and severity of infections; clinical presentation of infections may also be atypical.

Unless they have had chickenpox, patients receiving oral or parenteral corticosteroids for purposes other than replacement should be regarded as being at risk of severe chickenpox. Manifestations of fulminant illness include pneumonia, hepatitis and disseminated intravascular coagulation; rash is not necessarily a prominent feature. Passive immunisation with varicella–zoster immunoglobulin is needed for exposed non–immune patients receiving systemic corticosteroids or for those who have used them within the previous 3 months. Confirmed chickenpox warrants specialist care and urgent treatment. Corticosteroids should not be stopped and dosage may need to be increased.

Patients taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Hydrocortisone

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Prednisolone

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Dexamethasone

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is sufficient to inhibit corticotropin secretion for 24 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

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Pharmacology: Respiratory and Endocrine

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Regarding the management of diabetic ketoacidosis (DKA), once the blood-glucose concentration falls below 14 mmol/L, glucose should be given alongside insulin as:

a 10% glucose intravenous infusion at a rate of 125 mL/hour

b 20% glucose intravenous infusion at a rate of 125 mL/hour

c 10% glucose intravenous infusion at a rate of 250 mL/hour

d 5% glucose intravenous infusion at a rate of 125 mL/hour

e 5% glucose intravenous infusion at a rate of 250 mL/hour

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Pharmacology: Respiratory and Endocrine



Question 49 of 76

Regarding the management of diabetic ketoacidosis (DKA), once the blood-glucose concentration falls below 14 mmol/L, glucose should be given alongside insulin as:

a) 10% glucose intravenous infusion at a rate of 125 mL/hour ✔

b) 20% glucose intravenous infusion at a rate of 125 mL/hour

c) 10% glucose intravenous infusion at a rate of 250 mL/hour

d) 5% glucose intravenous infusion at a rate of 125 mL/hour

e) 5% glucose intravenous infusion at a rate of 250 mL/hour

Answer

Once blood-glucose concentration falls below 14 mmol/litre, glucose 10% should be given by intravenous infusion (into a large vein through a large-gauge needle) at a rate of 125 mL/hour, in addition to the sodium chloride 0.9% infusion.

Notes

Diabetic ketoacidosis (DKA) consists of the biochemical triad of ketonaemia (ketosis), hyperglycaemia, and acidaemia.

Pathophysiology

DKA usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones (i.e. glucagon, cortisol, growth hormone, catecholamines). This type of hormonal imbalance enhances hepatic gluconeogenesis and glycogenolysis resulting in severe hyperglycaemia. Enhanced lipolysis increases serum free fatty acids that are then metabolised as an alternative energy source in the process of ketogenesis. This results in accumulation of large quantities of ketone bodies and subsequent metabolic acidosis. Fluid depletion occurs due to osmotic diuresis secondary to hyperglycaemia, vomiting, and inability to take in fluid due to a diminished level of consciousness.

Diagnosis

- Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
- Blood glucose > 11.0mmol/L or known diabetes mellitus
- Bicarbonate (HCO3-) < 15.0mmol/L and/or venous pH < 7.3

Management of diabetic ketoacidosis in adults

- Intravenous fluids
  - If SBP is below 90 mmHg (adjusted for age, sex, and medication as appropriate), 500 mL sodium chloride 0.9% should be given by intravenous infusion over 10 – 15 minutes, and repeated if SBP still < 90 mmHg
  - When SBP is greater than 90 mmHg, sodium chloride infusion should be continued at a rate that replaces deficit and provides maintenance.
- Potassium replacement
  - Potassium chloride (40 mmol/L) should be included in the fluids (as long as the serum potassium level < 5.5 mmol/L and the patient is passing urine), and the plasma potassium concentration maintained between 3.5 – 5.5 mmol/L (measured at 60 minutes, 2 hours, and 2 hourly thereafter; and hourly if outside the normal range).
- Insulin
  - An intravenous insulin infusion should be started at a concentration of 1 unit/mL, at a fixed rate of 0.1 units/kg/hour.
  - Established subcutaneous long-acting insulin therapy should be continued concomitantly.
  - Blood ketone and blood glucose concentrations should be checked hourly and the insulin infusion rate adjusted accordingly. Blood ketone concentration should fall by at least 0.5 mmol/litre/hour and blood glucose concentration should fall by at least 3 mmol/litre/hour.
- Glucose
  - Once blood-glucose concentration falls below 14 mmol/litre, glucose 10% should be given by intravenous infusion (into a large vein through a large-gauge needle) at a rate of 125 mL/hour, in addition to the sodium chloride 0.9% infusion.

The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

The management of hyperosmolar hyperglycaemic state or hyperosmolar hyperglycaemic nonketotic coma is similar to that of diabetic ketoacidosis, although lower rates of insulin infusion are usually necessary and slower rehydration may be required.

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Pharmacology: Respiratory and Endocrine

Question 50 of 76

Regarding theophylline, which of the following statements is INCORRECT:

a There is an increased risk of convulsions when theophylline is given with quinolones.

b Theophylline in overdose may cause severe and intractable vomiting.

c Both the frequency and severity of adverse effects increase at plasma-theophylline concentrations above 20 mg/litre.

d Theophylline is associated with an increased risk of tendon damage, including rupture.

e Theophylline should not be prescribed to people with porphyria.

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Pharmacology: Respiratory and Endocrine

Question 50 of 76

Regarding theophylline, which of the following statements is INCORRECT:

a) ✖
There is an increased risk of convulsions when theophylline is given with quinolones.

b) Theophylline in overdose may cause severe and intractable vomiting.

c)
Both the frequency and severity of adverse effects increase at plasma-theophylline concentrations above 20 mg/litre.

d) Theophylline is associated with an increased risk of tendon damage, including rupture. ✔

e) Theophylline should not be prescribed to people with porphyria.

Answer

Theophylline is not recognised as a cause of tendon damage or rupture, unlike ciprofloxacin. The remaining statements listed are correct.

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

Question 51 of 76

Which of the following corticosteroids has the most potent mineralocorticoid effect:

a Betamethasone

b Fludrocortisone

c Hydrocortisone

d Methylprednisolone

e Dexamethasone

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Pharmacology: Respiratory and Endocrine

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Which of the following corticosteroids has the most potent mineralocorticoid effect:

a) Betamethasone
b) Fludrocortisone ✔
c) Hydrocortisone
d) Methylprednisolone
e) **Dexamethasone** ✖

Answer

Fludrocortisone has the most potent mineralocorticosteroid activity, making it ideal for mineralocorticoid replacement in adrenal insufficiency.

Notes

In comparing the relative potencies of corticosteroids in terms of their anti-inflammatory effects, it is worth noting that high glucocorticoid activity in itself is of no advantage unless it is accompanied by relatively low mineralocorticoid activity.

Anti-inflammatory potency

Equivalent anti-inflammatory doses of corticosteroids:

Prednisolone 5 mg = Dexamethasone 750 micrograms = Hydrocortisone 20 mg

This does not take into account their mineralocorticoid effects, nor their duration of action.

Side effects

Overdosage or prolonged use can exaggerate some of the normal physiological actions of corticosteroids leading to mineralocorticoid and glucocorticoid side effects. Mineralocorticoid side effects are most marked with fludrocortisone, but are significant with hydrocortisone, negligible with the high potency glucocorticoids, betamethasone and dexamethasone, and occur only slightly with methylprednisolone and prednisolone.

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Side effects can be minimised by using the lowest effective dose for the minimum period possible. The suppressive action of a corticosteroid on cortisol secretion is least when it is given as a single dose in the morning. Whenever possible local treatment with creams, intra-articular injections, inhalations, eye drops, or enemas should be used in preference to systemic treatment.

Adrenal suppression

During prolonged therapy with corticosteroids, particularly with systemic use, adrenal atrophy develops and can persist for years after stopping. Abrupt withdrawal after a prolonged period can lead to acute adrenal insufficiency, hypotension, or death. Patients removed from long-term glucocorticoid therapy must be weaned off the drug over several days, using progressively lower doses to allow recovery of adrenal responsiveness.

To compensate for a diminished adrenocortical response caused by prolonged corticosteroid treatment, any significant intercurrent illness, trauma, or surgical procedure requires a temporary increase in corticosteroid dose, or if already stopped, a temporary reintroduction of corticosteroid treatment.

Patients on long-term corticosteroid treatment should carry a steroid treatment card which gives guidance on minimising risk and provides details of prescriber, drug, dosage and duration of treatment.

Infections

Prolonged courses of corticosteroids increase susceptibility to infections and severity of infections; clinical presentation of infections may also be atypical.

Unless they have had chickenpox, patients receiving oral or parenteral corticosteroids for purposes other than replacement should be regarded as being at risk of severe chickenpox. Manifestations of fulminant illness include pneumonia, hepatitis and disseminated intravascular coagulation; rash is not necessarily a prominent feature. Passive immunisation with varicella–zoster immunoglobulin is needed for exposed non–immune patients receiving systemic corticosteroids or for those who have used them within the previous 3 months. Confirmed chickenpox warrants specialist care and urgent treatment. Corticosteroids should not be stopped and dosage may need to be increased.

Patients taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Hydrocortisone

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Prednisolone

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Dexamethasone

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is suffcient to inhibit corticotropin secretion for 2 4 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

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Pharmacology: Respiratory and Endocrine

Question 52 of 76

Regarding inhaled corticosteroids, which of the following statements is CORRECT:

a Symptom improvement usually occurs about 12 hours after initiation of treatment.

b Inhaled corticosteroids do not cause systemic side effects.

c They should be taken as and when required for worsening of asthma.

d Inhaled corticosteroids should be given at a higher dose during an acute attack of asthma.

e The mouth should be rinsed out with water after using an inhaled corticosteroid.

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Pharmacology: Respiratory and Endocrine

Question 52 of 76

Regarding inhaled corticosteroids, which of the following statements is CORRECT:

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Symptom improvement usually occurs about 12 hours after initiation of treatment.
b) Inhaled corticosteroids do not cause systemic side effects.
c) They should be taken as and when required for worsening of asthma.
d) Inhaled corticosteroids should be given at a higher dose during an acute attack of asthma.
e) The mouth should be rinsed out with water after using an inhaled corticosteroid. ✔

Answer

Inhaled corticosteroids can cause oral candidiasis and patients should be advised to rinse their mouth out with water after using the inhaler. Systemic corticosteroids, not increased inhaled corticosteroids should be given during an acute attack of asthma. Inhaled corticosteroids must be used regularly for maximum benefit, not as and when; alleviation of symptoms usually occurs 3 to 7 days after initiation. Systemic adverse effects may occur rarely, particularly if high doses are prescribed for long periods of time or in concomitant use of other corticosteroid preparations.

Notes

Corticosteroids reduce airway inflammation and hence oedema and mucus secretion. Regular use of inhaled corticosteroids (e.g. beclomethasone) reduces the risk of exacerbation of asthma.

An inhaled corticosteroid is used regularly for prophylaxis of asthma when patients require a beta-2 agonist more than twice a week, or if symptoms disturb sleep at least once a week, or if the patient has suffered an exacerbation in the last 2 years requiring a systemic corticosteroid. Corticosteroid inhalers must be used regularly for maximum benefit; alleviation of symptoms usually occurs 3 to 7 days after initiation.

Current and previous smoking reduces the effectiveness of inhaled corticosteroids and higher doses may be necessary.

Contraindications

There are no contraindications to the use of inhaled corticosteroids.

Inhaled corticosteroids should be used with caution in people with tuberculosis (potential for exacerbation or reactivation) or in untreated systemic fungal, bacterial, parasitic or viral infection.

Adverse effects

Local adverse effects of inhaled corticosteroids include:

- Oral candidiasis, sore mouth, dysphonia, hoarseness (patients should be advised to rinse their mouth with water after inhalation)
- Paradoxical bronchospasm (very rare and usually mild)

Systemic adverse effects may occur rarely, particularly if high doses are prescribed for long periods of time or in concomitant use of other corticosteroid preparations.

Systemic effects include:

- Reduced bone mineral density predisposing the person to osteoporosis
- Bruising, cataract, and glaucoma
- Adrenal suppression , adrenal crisis, coma, and death — very rarely, this has been reported in children taking long-term inhaled corticosteroids
- Psychological and behavioural changes (such as psychomotor hyperactivity, sleep disorders, anxiety, depression, and aggression)
- Growth suppression in children – this does not seem to occur with recommended doses of inhaled corticosteroids

Systemic corticosteroid therapy in asthma

Systemic corticosteroid therapy may be necessary during episodes of stress, such as severe infection, or if the asthma is worsening, when higher doses are needed and access of inhaled drug to small airways may be reduced; patients may need a reserve supply of corticosteroid tablets.

An acute attack of asthma should be treated with a short course of an oral corticosteroid (e.g. prednisolone) or intravenous corticosteroid (e.g. hydrocortisone) if oral intake is not possible, starting with a high dose. An oral corticosteroid should normally be taken as a single dose in the morning to reduce the disturbance to circadian cortisol secretion.

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Pharmacology: Respiratory and Endocrine

Question 53 of 76

Plasma theophylline concentration is decreased by which one of the following:

a Hepatic impairment

b Obesity

c Alcohol consumption

d Heart failure

e Fever

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Pharmacology: Respiratory and Endocrine



Question 53 of 76

Plasma theophylline concentration is decreased by which one of the following:

a) **Hepatic impairment** ✗
b) Obesity
c) Alcohol consumption ✓
d) Heart failure
e) Fever

Answer

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

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Regarding the management of acute asthma in adults, which of the following statements is INCORRECT:

a Routine prescription of antibiotics is not indicated for patients with acute asthma.

b Intravenous aminophylline has been shown to result in significant additional bronchodilation compared to standard care.

c Continuous salbutamol nebulisation should be considered in patients with severe acute asthma that is poorly responsive to initial bolus dose of salbutamol.

d The dose of magnesium sulphate in acute severe asthma is 1.2 – 2 g intravenous infusion over 20 minutes.

e Nebulised magnesium is not recommended for treatment in adults with acute asthma.

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Pharmacology: Respiratory and Endocrine

Question 54 of 76

Regarding the management of acute asthma in adults, which of the following statements is INCORRECT:

a) ✗
Routine prescription of antibiotics is not indicated for patients with acute asthma.
b) ✓
Intravenous aminophylline has been shown to result in significant additional bronchodilation compared to standard care.
c)
Continuous salbutamol nebulisation should be considered in patients with severe acute asthma that is poorly responsive to initial bolus dose of salbutamol.
d)
The dose of magnesium sulphate in acute severe asthma is 1.2 – 2 g intravenous infusion over 20 minutes.
e) Nebulised magnesium is not recommended for treatment in adults with acute asthma.

Answer

Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting. However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr).

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

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Regarding the management of diabetic ketoacidosis (DKA), if the systolic BP is initially < 90 mmHg, which of the following should be given:

a 500 mL sodium chloride 0.9% intravenous infusion over 10 – 15 minutes

b 500 mL colloid intravenous infusion over 10 – 15 minutes

c 1 L sodium chloride 0.9% intravenous infusion over 10 – 15 minutes

d 500 mL sodium chloride 0.9% intravenous infusion over 1 hour

e 500 mL sodium bicarbonate intravenous infusion over 10 – 15 minutes

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Pharmacology: Respiratory and Endocrine



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b) 500 mL colloid intravenous infusion over 10 – 15 minutes

c) 1 L sodium chloride 0.9% intravenous infusion over 10 – 15 minutes

d) 500 mL sodium chloride 0.9% intravenous infusion over 1 hour

e) 500 mL sodium bicarbonate intravenous infusion over 10 – 15 minutes

Answer

If SBP is below 90 mmHg (adjusted for age, sex, and medication as appropriate), 500 mL sodium chloride 0.9% should be given by intravenous infusion over 10 – 15 minutes, and repeated if SBP still < 90 mmHg. When SBP is greater than 90 mmHg, sodium chloride infusion should be continued at a rate that replaces deficit and provides maintenance.

Notes

Diabetic ketoacidosis (DKA) consists of the biochemical triad of ketonaemia (ketosis), hyperglycaemia, and acidaemia.

Pathophysiology

DKA usually occurs as a consequence of absolute or relative insulin deficiency that is accompanied by an increase in counter-regulatory hormones (i.e. glucagon, cortisol, growth hormone, catecholamines). This type of hormonal imbalance enhances hepatic gluconeogenesis and glycogenolysis resulting in severe hyperglycaemia. Enhanced lipolysis increases serum free fatty acids that are then metabolised as an alternative energy source in the process of ketogenesis. This results in accumulation of large quantities of ketone bodies and subsequent metabolic acidosis. Fluid depletion occurs due to osmotic diuresis secondary to hyperglycaemia, vomiting, and inability to take in fluid due to a diminished level of consciousness.

Diagnosis

- Ketonaemia > 3.0mmol/L or significant ketonuria (more than 2+ on standard urine sticks)
- Blood glucose > 11.0mmol/L or known diabetes mellitus
- Bicarbonate (HCO3-) < 15.0mmol/L and/or venous pH < 7.3

Management of diabetic ketoacidosis in adults

- Intravenous fluids
  - If SBP is below 90 mmHg (adjusted for age, sex, and medication as appropriate), 500 mL sodium chloride 0.9% should be given by intravenous infusion over 10 – 15 minutes, and repeated if SBP still < 90 mmHg.
  - When SBP is greater than 90 mmHg, sodium chloride infusion should be continued at a rate that replaces deficit and provides maintenance.
- Potassium replacement
  - Potassium chloride (40 mmol/L) should be included in the fluids (as long as the serum potassium level < 5.5 mmol/L and the patient is passing urine), and the plasma potassium concentration maintained between 3.5 – 5.5 mmol/L (measured at 60 minutes, 2 hours, and 2 hourly thereafter; and hourly if outside the normal range).
- Insulin
  - An intravenous insulin infusion should be started at a concentration of 1 unit/mL, at a fixed rate of 0.1 units/kg/hour.
  - Established subcutaneous long-acting insulin therapy should be continued concomitantly.
  - Blood ketone and blood glucose concentrations should be checked hourly and the insulin infusion rate adjusted accordingly. Blood ketone concentration should fall by at least 0.5 mmol/litre/hour and blood glucose concentration should fall by at least 3 mmol/litre/hour.
- Glucose
  - Once blood-glucose concentration falls below 14 mmol/litre, glucose 10% should be given by intravenous infusion (into a large vein through a large-gauge needle) at a rate of 125 mL/hour, in addition to the sodium chloride 0.9% infusion.

The insulin infusion should be continued until blood ketone concentration is below 0.3 mmol/litre, blood pH is above 7.3 and the patient is able to eat and drink; ideally the insulin infusion should be stopped about an hour after giving subcutaneous fast-acting insulin and a meal.

The management of hyperosmolar hyperglycaemic state or hyperosmolar hyperglycaemic nonketotic coma is similar to that of diabetic ketoacidosis, although lower rates of insulin infusion are usually necessary and slower rehydration may be required.

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Pharmacology: Respiratory and Endocrine

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Regarding antimuscarinic bronchodilators, which of the following statements is CORRECT:

a Inhaled antimuscarinics have significant systemic side effects.

b Short-acting β2 agonists are slower acting but more effective than antimuscarinics.

c Ipratropium bromide has a duration of action of 2 hours.

d Ipratropium bromide has its maximal effect after 5 – 10 minutes.

e There are no important drug interactions with inhaled muscarinic antagonists.

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Pharmacology: Respiratory and Endocrine



Question 56 of 76

Regarding antimuscarinic bronchodilators, which of the following statements is CORRECT:

a) **Inhaled antimuscarinics have significant systemic side effects.** ✖
b) Short-acting β2 agonists are slower acting but more effective than antimuscarinics.
c) Ipratropium bromide has a duration of action of 2 hours.
d) Ipratropium bromide has its maximal effect after 5 – 10 minutes.
e) There are no important drug interactions with inhaled muscarinic antagonists. ✔

Answer

There are no important drug interactions with inhaled muscarinic antagonists. Inhaled antimuscarinics are generally well tolerated as they are poorly absorbed systemically. Ipratropium bromide has its maximal effect 30 – 60 minutes after use and its duration of action is 3 to 6 hours. Ipratropium bromide can provide short-term relief in chronic asthma, but short-acting beta-2 agonists act more quickly and are preferred. Ipratropium bromide by nebulisation can be added to other standard treatment in life-threatening asthma or if acute asthma fails to improve with standard therapy.

Notes

Ipratropium bromide, a short-acting antimuscarinic bronchodilator, causes bronchodilation by blocking the cholinergic nerves in the airways.

Ipratropium bromide can provide short-term relief in chronic asthma, but short-acting beta-2 agonists act more quickly and are preferred. Ipratropium bromide by nebulisation can be added to other standard treatment in life-threatening asthma or if acute asthma fails to improve with standard therapy.

The aerosol inhalation of ipratropium bromide may be used for short-term relief in mild COPD in patients who are not using a long-acting antimuscarinic drug. Its maximal effect occurs 30 – 60 minutes after use; its duration of action is 3 to 6 hours and bronchodilation can usually be maintained with treatment 3 times a day.

Cautions

Ipratropium bromide should be used with caution in:

- Men with prostatic hyperplasia and bladder-outflow obstruction (worsened urinary retention has been reported in elderly men)
- People with chronic kidney disease (CKD) stages 3 and above (because of the risk of drug toxicity)
- People with angle-closure glaucoma (nebulised mist of antimuscarinic drugs can precipitate or worsen acute angle-closure glaucoma)

Interactions

There are no important drug interactions with inhaled muscarinic antagonists.

Side effects

Inhaled antimuscarinics are generally well tolerated as they are poorly absorbed systemically.

Their adverse effects include:

- Dry mouth and abnormal taste in the mouth
- Nasal congestion and dryness of nasal mucosa
- Acute angle-closure glaucoma (reported in people on nebulised ipratropium)

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Pharmacology: Respiratory and Endocrine

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Which of the following is a common adverse effect of glucagon:

a Hyperkalaemia

b Nausea and vomiting

c Tremor

d Tachycardia

e Hypoglycaemia

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Pharmacology: Respiratory and Endocrine

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Which of the following is a common adverse effect of glucagon:

a) **Hyperkalaemia** ✗
b) Nausea and vomiting ✓
c) Tremor
d) Tachycardia
e) Hypoglycaemia

Answer

Adverse effects include:

- Nausea and vomiting
- Hyperglycaemia
- Hypokalaemia
- Hypotension

Notes

Management of hypoglycaemia in adults

- In adults who are conscious, cooperative and can swallow:
  - Give 15 – 20 g quick acting carbohydrate of the patient's choice where possible e.g. 90 – 120 mL of Lucozade or 5 – 7 Dextrosol tablets
  - Repeat capillary blood glucose 10 – 15 minutes later
  - If blood glucose is still < 4.0 mmol/L, repeat step 1 (no more than 3 treatments in total)
  - If blood glucose remains < 4.0 mmol/L after 45 minutes or 3 cycles, consider:
    - 1 mg glucagon IM
    - IV 10% glucose infusion at 100ml/hr
- In adults who are conscious but uncooperative:
  - Give either 1.5 – 2 tubes Glucogel/Dextrogel (may repeat up to 3 times)
  - If this is ineffective give glucagon 1 mg IM (may only give once)
  - If blood glucose level remains less than 4.0 mmol/L after 45 minutes (or 3 cycles), consider IV 10% glucose infusion at 100ml/hr
- In adults who are unconscious:
  - Give either:
    - 1 mg glucagon intramuscularly (if not effective after 10 – 15 minutes, IV glucose should be given)
    - 75 – 80 ml of 20% glucose intravenously over 10 – 15 minutes
    - 150 – 160 ml of 10% glucose intravenously over 10 – 15 minutes

Once blood glucose is > 4.0 mmol/L and the patient recovered, give a long acting carbohydrate of the patient's choice where possible e.g. two biscuits, one slice of bread. Note that patients given glucagon require a larger portion of long acting carbohydrate to replenish glycogen stores (double the suggested amount).

If the hypoglycaemia was due to sulfonylurea or long acting insulin therapy then be aware that the risk of hypoglycaemia may persist for up to 24 – 36 hours following the last dose, especially if there is concurrent renal impairment.

Glucagon

Glucagon, a polypeptide hormone produced by the alpha cells of the islets of Langerhans, increases plasma-glucose by mobilising glycogen stored in the liver. Glycogen promotes glycogenolysis and gluconeogenesis.

Glucagon may take up to 15 minutes to have an effect and will be less effective in alcoholics, prolonged starvation and severe liver disease when glycogen stores are depleted. In this situation or if prolonged treatment is required, IV glucose is better. Glucagon may also be less effective in patients prescribed sulfonylurea therapy.

Glucagon may also be used as an antidote in beta-blocker overdose and in anaphylaxis in patients on beta-blockers that fail to respond to adrenaline.

Glucagon is contraindicated in pheochromocytoma.

Adverse effects include:

- Nausea and vomiting
- Hyperglycaemia
- Hypokalaemia
- Hypotension

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Pharmacology: Respiratory and Endocrine

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A 5 year old child, with a known latex allergy, accidentally comes into contact with some latex gloves. She has significant lip and tongue swelling, and signs of impending airway obstruction. What is the most appropriate dose of adrenaline for this patient:

a 0.05 grams intramuscularly

b 0.2 grams intramuscularly

c 500 micrograms intramuscularly

d 300 micrograms intramuscularly

e 150 micrograms intramuscularly

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a) 0.05 grams intramuscularly
b) 0.2 grams intramuscularly
c) **500 micrograms intramuscularly** ✘
d) 300 micrograms intramuscularly
e) 150 micrograms intramuscularly ✔

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Answer

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
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Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

Question 59 of 76

A 14 year old patient, with a history of asthma and atopy, is given penicillin in hospital. He immediately develops an itchy rash, angioedema and worsening stridor and wheeze. What is the most appropriate dose of adrenaline for this patient:

a 5 milligrams intramuscularly

b 0.5 grams intramuscularly

c 500 micrograms intramuscularly

d 300 micrograms intramuscularly

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Pharmacology: Respiratory and Endocrine

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Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

Question 60 of 76

A 24 year old patient with life-threatening acute asthma is too unwell to take oral steroids. What is the most appropriate alternative to oral steroids in this patient:

a Intravenous hydrocortisone 100 mg daily

b Intravenous hydrocortisone 200 mg daily

c Intravenous hydrocortisone 300 mg daily

d Intravenous hydrocortisone 400 mg daily

e Intravenous hydrocortisone 500 mg daily

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b) **Intravenous hydrocortisone 200 mg daily** ✗
c) Intravenous hydrocortisone 300 mg daily
d) Intravenous hydrocortisone 400 mg daily ✓
e) Intravenous hydrocortisone 500 mg daily

Answer

The BTS/SIGN guidelines state that steroids in adequate doses should be given to all patients with an acute asthma attack. Prednisolone 40 – 50 mg should be prescribed daily for at least 5 days or until recovery. Parenteral hydrocortisone (400 mg daily, 100 mg QDS) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

Question 61 of 76

The second generation antihistamine, cetirizine is a less-sedating antihistamine than the older antihistamine, chlorphenamine because:

a It is selective for H1-receptors.

b It is cleared more rapidly by the kidneys.

c It is metabolised more quickly by the liver.

d It is less lipid-soluble so less able to cross the blood brain barrier.

e It has less anticholinergic effect.

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Pharmacology: Respiratory and Endocrine

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The second generation antihistamine, cetirizine is a less-sedating antihistamine than the older antihistamine, chlorphenamine because:

a) **It is selective for H1-receptors.** ✗
b) It is cleared more rapidly by the kidneys.
c) It is metabolised more quickly by the liver.
d) It is less lipid-soluble so less able to cross the blood brain barrier. ✓
e) It has less anticholinergic effect.

Answer

All older antihistamines such as chlorphenamine cause sedation. The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent.

Notes

Antihistamines are competitive inhibitors at the H1-receptor (in contrast to H2 receptor antagonists used to decrease gastric acid secretion). They act to relax histamine-induced bronchoconstriction, block the vasodilator effect of histamine, inhibit histamine-induced increases in capillary permeability and block mucus secretion and sensory nerve stimulation.

Histamine (H1)-receptor antagonists are well absorbed after oral administration. The effects of these agents are usually seen in 30 minutes (with maximal effects at 1 – 2 h); the duration of action is 3 – 8 hours for first-generation compounds and 3 – 24 hours for second-generation compounds.

H1-receptor antagonists are metabolised in the liver; many induce microsomal enzymes and alter their own metabolism and that of other drugs.

Indications

- Allergic rhinitis and conjunctivitis
- Urticarial rashes, pruritus, insect bites and stings
- Angioedema
- Anaphylaxis (second line adjunct to adrenaline)
- Nausea/vomiting and prevention of motion sickness
- Insomnia

Chlorphenamine

All older antihistamines cause sedation but alimemazine tartrate and promethazine may be more sedating whereas chlorphenamine maleate and cyclizine may be less so. This sedating activity is sometimes used to manage the pruritus associated with some allergies or used to manage occasional insomnia. There is little evidence that any one of the older, 'sedating' antihistamines is superior to another and patients vary widely in their response.

Cetirizine

The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent.

Cautions

Antihistamines should usually be avoided in acute porphyrias (although some antihistamines are thought to be safe).

Antihistamines should be used with caution in epilepsy, prostatic hypertrophy, urinary hypertension, hepatic impairment and susceptibility to angle-closure glaucoma.

Side effects (significantly reduced with second-generation agents)

Elderly patients and children are more susceptible to side effects.

Common side effects of antihistamines may include:

- Anticholinergic effects (blurred vision, dry mouth, urinary retention)
- Headache
- Gastrointestinal disturbances
- Psychomotor impairment (sedation, dizziness and loss of appetite)

Drowsiness may affect performance of skilled tasks (e.g. cycling or driving); sedating effects are enhanced by alcohol and opioid analgesics.

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Pharmacology: Respiratory and Endocrine

Question 62 of 76

Regarding theophylline, which of the following statements is INCORRECT:

a Excretion of lithium may be potentiated by concomitant therapy with theophylline.

b It is metabolised in the liver.

c It has a narrow therapeutic index.

d Theophylline significantly increases the anticoagulant effect of warfarin.

e The risk of hypokalaemia is increased if given concomitantly with beta-2 agonists.

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Pharmacology: Respiratory and Endocrine



Question 62 of 76

Regarding theophylline, which of the following statements is INCORRECT:

a) ✖
Excretion of lithium may be potentiated by concomitant therapy with theophylline.
b) It is metabolised in the liver.
c) It has a narrow therapeutic index.
d) Theophylline significantly increases the anticoagulant effect of warfarin. ✔
e) The risk of hypokalaemia is increased if given concomitantly with beta-2 agonists.

Answer

Theophylline is not noted to interact with warfarin in the BNF. The remaining statements listed are correct.

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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Pharmacology: Respiratory and Endocrine

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Plasma theophylline concentration is increased by all of the following EXCEPT for:

a Heart failure

b Hepatic impairment

c Viral infections

d Elderly

e Smoking

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a) Heart failure
b) Hepatic impairment
c) **Viral infections** ✖
d) Elderly
e) Smoking ✔

Answer

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

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Pharmacology: Respiratory and Endocrine

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Regarding oxygen therapy, which of the following statements is CORRECT:

a. In acutely ill patients, oxygen saturations should always be maintained at > 98%.

b. For patients at risk of hypercapnic respiratory failure, oxygen saturations should be maintained at 92 – 96%.

c. For patients at risk of hypercapnic respiratory failure, initial oxygen should be given at a concentration of 42% and titrated.

d. In carbon monoxide poisoning, high flow oxygen therapy should be continued regardless of oxygen saturations.

e. Long-term oxygen therapy may treat symptoms but has no effect on survival in COPD patients.

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Pharmacology: Respiratory and Endocrine

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For patients at risk of hypercapnic respiratory failure, oxygen saturations should be maintained at 92 – 96%.

c)
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d) ✔
In carbon monoxide poisoning, high flow oxygen therapy should be continued regardless of oxygen saturations.

e) Long-term oxygen therapy may treat symptoms but has no effect on survival in COPD patients.

Answer

In carbon monoxide poisoning high flow oxygen therapy should be continued regardless of oxygen saturations. High concentration oxygen promotes dissociation of carbon monoxide from carboxyhemoglobin and therapy should continue until carboxyhaemoglobin levels decrease to less than 10% and patients are asymptomatic. In most acutely ill patients with a normal or low arterial carbon dioxide (PaCO2), oxygen saturation should be 94 – 98% oxygen saturation. A lower target of 88 – 92% oxygen saturation is indicated for patients at risk of hypercapnic respiratory failure e.g. patients with COPD. Until blood gases can be measured, initial oxygen should be given using a controlled concentration of 28% or less, titrated towards the SpO2 of 88 – 92%. Long-term administration of oxygen (usually at least 15 hours daily) prolongs survival in some patients with COPD.

Notes

Oxygen should be regarded as a drug. It is prescribed for hypoxaemic patients to increase alveolar oxygen tension and decrease the work of breathing. The concentration of oxygen required depends on the condition being treated; the administration of an inappropriate concentration of oxygen can have serious or even fatal consequences.

High concentration oxygen therapy

In most acutely ill patients with a normal or low arterial carbon dioxide ($PaCO_2$), oxygen saturation should be 94 – 98% oxygen saturation. However, in some clinical situations such as cardiac arrest and carbon monoxide poisoning it is more appropriate to aim for the highest possible oxygen saturation until the patient is stable.

Low concentration oxygen therapy

A lower target of 88 – 92% oxygen saturation is indicated for patients at risk of hypercapnic respiratory failure e.g. patients with COPD. Until blood gases can be measured, initial oxygen should be given using a controlled concentration of 28% or less, titrated towards the SpO2 of 88 – 92%. The aim is to provide the patient with enough oxygen to achieve an acceptable arterial oxygen tension without worsening carbon dioxide retention and respiratory acidosis.

Long term oxygen therapy

Long-term administration of oxygen (usually at least 15 hours daily) prolongs survival in some patients with COPD. Assessment for long-term oxygen therapy requires measurement of arterial blood gas tensions. A nasal cannula is usually preferred for long-term oxygen therapy from an oxygen concentrator. It can, however, produce dermatitis and mucosal drying in sensitive individuals.

Giving oxygen by nasal cannula allows the patient to talk, eat, and drink, but the concentration of oxygen is not controlled; this may not be appropriate for acute respiratory failure. Increased respiratory depression is seldom a problem in patients with stable respiratory failure treated with low concentrations of oxygen although it may occur during exacerbations; patients and relatives should be warned to call for medical help if drowsiness or confusion occur.

Intermittent oxygen therapy

Oxygen is occasionally prescribed for short-burst (intermittent) use for episodes of breathlessness not relieved by other treatment in patients with severe chronic obstructive pulmonary disease, interstitial lung disease, heart failure, and in palliative care.

Ambulatory oxygen

Ambulatory oxygen is prescribed for patients on long-term oxygen therapy who need to be away from home on a regular basis. Patients who are not on long-term oxygen therapy can be considered for ambulatory oxygen therapy if there is evidence of exercise-induced oxygen desaturation and of improvement in blood oxygen saturation and exercise capacity with oxygen.

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Pharmacology: Respiratory and Endocrine

Question 65 of 76

Adrenaline acts on which of the following receptors:

a α receptors

b β1 receptors

c β1 and β2 receptors

d α, β1 and β2 receptors

e β2 receptors

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Pharmacology: Respiratory and Endocrine

Question 65 of 76

Adrenaline acts on which of the following receptors:

a) α receptors
b) β1 receptors
c) β1 and β2 receptors
d) **α, β1 and β2 receptors** ✔
e) β2 receptors

Answer

Adrenaline is a catecholamine that acts on both alpha and beta receptors and increases both heart rate and contractility (beta1 effects); it can cause peripheral vasodilation (a beta2 effect) or vasoconstriction (an alpha effect).

Notes

Adrenaline is a catecholamine that acts on both alpha and beta receptors and increases both heart rate and contractility (beta1 effects); it can cause peripheral vasodilation (a beta2 effect) or vasoconstriction (an alpha effect).

Adrenaline is used in cardiopulmonary resuscitation, in the emergency management of acute allergic and anaphylactic reactions and in the management of severe croup (as a nebulised solution).

Mechanism of action in anaphylaxis

As an alpha-receptor agonist, adrenaline reverses peripheral vasodilation and increased vascular permeability reducing hypotension and oedema.

As a beta-receptor agonist it dilates bronchial airways, increases the force and rate of myocardial contraction, and suppresses histamine and leukotriene release.

Adrenaline also alleviates pruritus, urticaria, and angioedema and may relieve gastrointestinal and genitourinary symptoms associated with anaphylaxis because of its relaxer effects on the smooth muscle of the stomach, intestine, uterus and urinary bladder.

Adrenaline increases glycogenolysis, reduces glucose uptake by tissues, and inhibits insulin release in the pancreas, resulting in hyperglycemia and increased blood lactic acid.

Interactions

Severe anaphylaxis in patients taking beta-blockers may not respond to adrenaline—consider bronchodilator therapy. Furthermore, adrenaline can cause severe hypertension and bradycardia in those taking non-cardioselective beta-blockers.

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Pharmacology: Respiratory and Endocrine

Question 66 of 76

A 31 year old patient with a known nut allergy was eating at a Thai restaurant when he suddenly complained of lip and tongue swelling and difficulty breathing. He is brought to ED by ambulance with suspected anaphylaxis. What is the most appropriate first line treatment for this patient:

a 5 mg of adrenaline intramuscularly

b 0.5 mL of 1:10000 adrenaline solution intramuscularly

c 500 micrograms of adrenaline intramuscularly

d 0.5 mg of adrenaline intravenously if access has been achieved

e 0.5 mL of 1:1000 solution adrenaline intravenously if access has been achieved

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Pharmacology: Respiratory and Endocrine

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A 31 year old patient with a known nut allergy was eating at a Thai restaurant when he suddenly complained of lip and tongue swelling and difficulty breathing. He is brought to ED by ambulance with suspected anaphylaxis. What is the most appropriate first line treatment for this patient:

a) **5 mg of adrenaline intramuscularly** ✗
b) 0.5 mL of 1:10000 adrenaline solution intramuscularly
c) 500 micrograms of adrenaline intramuscularly ✓
d) 0.5 mg of adrenaline intravenously if access has been achieved
e) 0.5 mL of 1:1000 solution adrenaline intravenously if access has been achieved

Answer

Adrenaline should be given immediately if anaphylaxis is suspected at a dose of 500 micrograms (0.5 ml of 1:1000 solution). The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh. Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice.

Notes

Pathophysiology

Anaphylaxis is a severe, life-threatening, generalised type I hypersensitivity reaction. It occurs when an antigen binds to specific IgE immunoglobulins on mast cells triggering degranulation and release of inflammatory mediators (e.g. histamine, prostaglandins, and leukotrienes). Patients with pre-existing asthma, especially poorly controlled asthma, are at particular risk of life-threatening reactions.

Anaphylaxis can be triggered by any of a very broad range of triggers, but those most commonly identified include food, drugs, latex and venom. Of foods, nuts are the most common cause; muscle relaxants, antibiotics, NSAIDs and aspirin are the most commonly implicated drugs. Food is the commonest trigger in children and drugs the commonest in adults. A significant number of cases are idiopathic.

Clinical features

Anaphylaxis is characterised by sudden onset and rapidly developing, life-threatening airway, breathing and circulation problems associated with skin and/or mucosal changes.

- Airway
  - Pharyngeal or laryngeal oedema with hoarseness and stridor
- Breathing
  - Bronchospasm with tachypnoea, dyspnoea, wheeze, hypoxia, cyanosis, respiratory arrest
- Circulation
  - Peripheral vasodilation with hypotension, tachycardia, signs of peripheral shut-down (e.g. pale, clammy), shock, cardiac arrest
- Disability
  - Decreased brain perfusion leads to confusion, agitation and loss of consciousness
- Exposure
  - In about 80% of cases there are associated skin and mucosal changes such as flushing, pruritus, urticaria and angioedema; skin and/or mucosal changes can be absent or subtle in up to 20% of reactions
- Gastrointestinal symptoms
  - Nausea, vomiting, abdominal pain, diarrhoea, incontinence

Most reactions occur over several minutes; rarely, reactions may be slower in onset. The speed of onset of the reaction depends on the trigger e.g. intravenous medications will cause a more rapid onset than stings which in turn will cause a more rapid onset than ingestion of food.

Reactions can vary greatly, from hypotension alone, to reactions with predominantly asthmatic features, to cardiac/respiratory arrest. Skin or mucosal changes alone are not a sign of an anaphylactic reaction.

Immediate drug management

- **Oxygen**
  - Initially give 100% high flow oxygen using a mask with an oxygen reservoir
  - Once pulse oximetry is feasible target an SpO2 of 94 – 98%
- **Adrenaline** should be given immediately if anaphylaxis is suspected (see doses below)
  - As an alpha-receptor agonist, it reverses peripheral vasodilation and reduces oedema. As a beta-receptor agonist it dilates bronchial airways, increases the force of myocardial contraction, and suppresses histamine and leukotriene release
  - The intramuscular route is the best immediate route in anaphylaxis; the best site is the anterolateral aspect of the middle third of the thigh
  - Adrenaline should be given again after 5 minutes if there is no improvement, and repeat doses given at 5 minute intervals until there has been an adequate response
  - Patients taking tricyclic antidepressants, monoamine oxidase inhibitors or beta-blockers should be given only 50% of the normal dose
  - Consider IV/IM glucagon in patients who are not responding (especially in patients taking beta-blockers)
  - Intravenous adrenaline should only be administered by those experienced in the use and titration of vasopressors in their normal clinical practice
- **Intravenous fluids**
  - A rapid IV fluid challenge (20 mL/kg in a child or 500 – 1000 mL in an adult) should be administered and the response monitored; further doses can be given if necessary
  - There is no evidence to support the use of colloid over crystalloid fluid in resuscitation, therefore crystalloid fluid should be given as colloids have a higher risk of hypersensitivity reactions
- Second line drugs
  - Intravenous/intramuscular **chlorphenamine and hydrocortisone** should be given as second line drugs to reduce the severity and duration of symptoms (see doses below)
- **Bronchodilator therapy** for continuing respiratory deterioration
  - Inhaled or intravenous salbutamol
  - Inhaled ipratropium bromide
  - Intravenous aminophylline
  - Intravenous magnesium sulphate

Age group	IM adrenaline (1:1000)	IM/IV chlorphenamine	IM/IV hydrocortisone
Adult	500 mcg (0.5 mL)	10 mg	200 mg
Child > 12 years	500 mcg (0.5 mL)	10 mg	200 mg
Child 6 – 12 years	300 mcg (0.3 mL)	5 mg	100 mg
Child 6 months – 6 years	150 mcg (0.15 mL)	2.5 mg	50 mg
Child < 6 months	150 mcg (0.15 mL)	250 mcg/kg	25 mg

Confirmation of diagnosis

Emergency treatment should not be delayed and should be based on a clinical diagnosis of anaphylaxis.

Where diagnosis is difficult serum tryptase taken around the time of reaction may be helpful in subsequent investigation. Tryptase is a major component of mast cell granules, therefore in anaphylaxis mast cell degranulation leads to increased blood levels of tryptase. Tryptase concentration in the blood may not increase significantly until 30 minutes or more after the onset of symptoms and peaks 1 – 2 hours after onset. Ideally three timed samples are taken, the initial sample as soon as feasible after resuscitation has started, the second sample 1 – 2 hours after the start of the symptoms and the third sample either at 24 h or in convalescence (for baseline levels). Not all patients with anaphylaxis will have a documented rise, and therefore the absence of a rise does not reliably exclude the diagnosis.

Patient disposal

After intial treatment, patients should be observed for at least 6 hours in a clinical area with facilities for treating life-threatening ABC problems. They should then be reviewed by a senior clinician and a decision made about the need for further treatment or a longer period of observation. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 hours. This caution is particularly applicable to:

- severe reactions with slow onset caused by idiopathic anaphylaxis
- reactions in patients with severe asthma
- reactions with the possibility of continuing absorption of the trigger
- patients with a previous history of biphasic reactions
- patients who may not be able to respond to a deterioration appropriately (e.g. at night, live alone, live in area where access to emergency care is difficult etc.)

Before discharge from hospital all patients must be:

- Reviewed by a senior clinician
- Given clear instructions to return to hospital if symptoms return
- Considered for antihistamines and oral steroid therapy for up to 3 days (this is helpful for treatment of urticaria and may decrease the chance of further reaction)
- Considered for an adrenaline auto-injector or given a replacement
- Have a plan for follow-up, including contact with the patient's general practitioner

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Pharmacology: Respiratory and Endocrine

Question 67 of 76

Regarding steroids in the management of acute asthma in adults, which of the following statements is INCORRECT:

a Steroids should be given in adequate doses to all patients with an acute asthma attack.

b The earlier steroids are given in the acute attack, the better the outcome.

c Intravenous steroids are more effective at reducing relapses than oral steroids.

d Oral steroids should be continued daily for at least 5 days following the acute attack.

e At the end of the course of oral steroids, they can usually be stopped abruptly without tapering the dose.

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Pharmacology: Respiratory and Endocrine

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Regarding steroids in the management of acute asthma in adults, which of the following statements is INCORRECT:

a)
Steroids should be given in adequate doses to all patients with an acute asthma attack.
b) The earlier steroids are given in the acute attack, the better the outcome.
c) Intravenous steroids are more effective at reducing relapses than oral steroids. ✔
d) Oral steroids should be continued daily for at least 5 days following the acute attack.
e)
At the end of the course of oral steroids, they can usually be stopped abruptly without tapering the dose.

Answer

The BTS/SIGN guidelines state that steroids should be given in adequate doses to all patients with an acute asthma attack. Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome. Steroid tablets are as effective as injected steroids, provided they can be swallowed and retained. Prednisolone (40 – 50 mg daily) should be continued for at least five days or until recovery; at the point the steroid can be stopped abruptly without tapering the dose.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

Question 68 of 76

Regarding antihistamines, which of the following statements is CORRECT:

a The newer antihistamines have more of a sedating effect than older antihistamines.

b Antihistamines act on H2 receptors.

c Elderly patients and children are more susceptible to side effects.

d Cetirizine is an older antihistamine.

e Antihistamines are used as the first line treatment in anaphylaxis.

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Pharmacology: Respiratory and Endocrine



Question 68 of 76

Regarding antihistamines, which of the following statements is CORRECT:

a) ✖
The newer antihistamines have more of a sedating effect than older antihistamines.
b) Antihistamines act on H2 receptors.
c) Elderly patients and children are more susceptible to side effects. ✔
d) Cetirizine is an older antihistamine.
e) Antihistamines are used as the first line treatment in anaphylaxis.

Answer

Elderly patients and children are more susceptible to side effects. Antihistamines are competitive inhibitors at the H1-receptor. The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent. Antihistamines are used as a second line adjunct to adrenaline in anaphylaxis.

Notes

Antihistamines are competitive inhibitors at the H1-receptor (in contrast to H2 receptor antagonists used to decrease gastric acid secretion). They act to relax histamine-induced bronchoconstriction, block the vasodilator effect of histamine, inhibit histamine-induced increases in capillary permeability and block mucus secretion and sensory nerve stimulation.

Histamine (H1)-receptor antagonists are well absorbed after oral administration. The effects of these agents are usually seen in 30 minutes (with maximal effects at 1 – 2 h); the duration of action is 3 – 8 hours for first-generation compounds and 3 – 24 hours for second-generation compounds.

H1-receptor antagonists are metabolised in the liver; many induce microsomal enzymes and alter their own metabolism and that of other drugs.

Indications

- Allergic rhinitis and conjunctivitis
- Urticarial rashes, pruritus, insect bites and stings
- Angioedema
- Anaphylaxis (second line adjunct to adrenaline)
- Nausea/vomiting and prevention of motion sickness
- Insomnia

Chlorphenamine

All older antihistamines cause sedation but alimemazine tartrate and promethazine may be more sedating whereas chlorphenamine maleate and cyclizine may be less so. This sedating activity is sometimes used to manage the pruritus associated with some allergies or used to manage occasional insomnia. There is little evidence that any one of the older, 'sedating' antihistamines is superior to another and patients vary widely in their response.

Cetirizine

The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent.

Cautions

Antihistamines should usually be avoided in acute porphyrias (although some antihistamines are thought to be safe).

Antihistamines should be used with caution in epilepsy, prostatic hypertrophy, urinary hypertension, hepatic impairment and susceptibility to angle-closure glaucoma.

Side effects (significantly reduced with second-generation agents)

Elderly patients and children are more susceptible to side effects.

Common side effects of antihistamines may include:

- Anticholinergic effects (blurred vision, dry mouth, urinary retention)
- Headache
- Gastrointestinal disturbances
- Psychomotor impairment (sedation, dizziness and loss of appetite)

Drowsiness may affect performance of skilled tasks (e.g. cycling or driving); sedating effects are enhanced by alcohol and opioid analgesics.

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Pharmacology: Respiratory and Endocrine

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Salbutamol should be used with caution in patients with which of the following:

a Hypothyroidism

b Prostatic hyperplasia

c History of tendon rupture

d Susceptibility to QT-interval prolongation

e Gout

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Pharmacology: Respiratory and Endocrine



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Salbutamol should be used with caution in patients with which of the following:

a) **Hypothyroidism** ✗
b) Prostatic hyperplasia
c) History of tendon rupture
d) Susceptibility to QT-interval prolongation ✓
e) Gout

Answer

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Notes

Selective beta-2 agonists act directly on beta-2 receptors, causing smooth muscle relaxation and dilation of the airways.

Mild to moderate symptoms of asthma respond rapidly to the inhalation of a selective short-acting beta2 agonist such as salbutamol or terbutaline sulfate. Short-acting beta-2 agonists have a rapid onset of action (15 minutes) and their effects last for up to 4 hours. Salbutamol or terbutaline sulfate can be given intravenously for severe or life-threatening acute asthma; patients should be carefully monitored and the dose adjusted according to response and heart rate.

Short acting beta-2 agonists are used for immediate relief of asthma symptoms, while some long-acting β2 agonists (e.g. salmeterol) are added to an inhaled corticosteroid in patients requiring prophylactic treatment.

Cautions

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Interactions

Hypokalaemia may be potentiated by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics. This in turn may predispose to toxicity in patients taking digoxin.

Side effects

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
- Hypokalaemia
- Cardiac arrhythmia and paradoxical bronchospasm (rare)
- Acute angle-closure glaucoma
- QT-interval prolongation

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Pharmacology: Respiratory and Endocrine

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Which of the following is NOT a common side effect of antihistamines:

a Blurred vision

b Dry mouth

c Gastrointestinal disturbance

d Urinary retention

e Tremor

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Pharmacology: Respiratory and Endocrine



Question 70 of 76

Which of the following is NOT a common side effect of antihistamines:

a) Blurred vision
b) Dry mouth
c) Gastrointestinal disturbance
d) Urinary retention
e) **Tremor** ✔

Answer

Common side effects of antihistamines may include:

- Anticholinergic effects (blurred vision, dry mouth, urinary retention)
- Headache
- Gastrointestinal disturbances
- Psychomotor impairment (sedation, dizziness and loss of appetite)

These side effects are significantly reduced with second-generation agents.

Notes

Antihistamines are competitive inhibitors at the H1-receptor (in contrast to H2 receptor antagonists used to decrease gastric acid secretion). They act to relax histamine-induced bronchoconstriction, block the vasodilator effect of histamine, inhibit histamine-induced increases in capillary permeability and block mucus secretion and sensory nerve stimulation.

Histamine (H1)-receptor antagonists are well absorbed after oral administration. The effects of these agents are usually seen in 30 minutes (with maximal effects at 1 – 2 h); the duration of action is 3 – 8 hours for first-generation compounds and 3 – 24 hours for second-generation compounds.

H1-receptor antagonists are metabolised in the liver; many induce microsomal enzymes and alter their own metabolism and that of other drugs.

Indications

- Allergic rhinitis and conjunctivitis
- Urticarial rashes, pruritus, insect bites and stings
- Angioedema
- Anaphylaxis (second line adjunct to adrenaline)
- Nausea/vomiting and prevention of motion sickness
- Insomnia

Chlorphenamine

All older antihistamines cause sedation but alimemazine tartrate and promethazine may be more sedating whereas chlorphenamine maleate and cyclizine may be less so. This sedating activity is sometimes used to manage the pruritus associated with some allergies or used to manage occasional insomnia. There is little evidence that any one of the older, 'sedating' antihistamines is superior to another and patients vary widely in their response.

Cetirizine

The newer antihistamines e.g. cetirizine cause less sedation and psychomotor impairment than the older antihistamines because they are much less lipid soluble and penetrate the blood brain barrier only to a slight extent.

Cautions

Antihistamines should usually be avoided in acute porphyrias (although some antihistamines are thought to be safe).

Antihistamines should be used with caution in epilepsy, prostatic hypertrophy, urinary hypertension, hepatic impairment and susceptibility to angle-closure glaucoma.

Side effects (significantly reduced with second-generation agents)

Elderly patients and children are more susceptible to side effects.

Common side effects of antihistamines may include:

- Anticholinergic effects (blurred vision, dry mouth, urinary retention)
- Headache
- Gastrointestinal disturbances
- Psychomotor impairment (sedation, dizziness and loss of appetite)

Drowsiness may affect performance of skilled tasks (e.g. cycling or driving); sedating effects are enhanced by alcohol and opioid analgesics.

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Pharmacology: Respiratory and Endocrine

Question 71 of 76

Regarding the management of acute asthma in adults, which of the following statements is INCORRECT:

a Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care.

b Parenteral hydrocortisone or intramuscular methylprednisolone are alternatives in patients who are unable to take oral prednisolone.

c Intravenous magnesium sulphate should be considered for patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy.

d High-dose inhaled short-acting beta2-agonists are the first line treatment for acute asthma.

e Nebulised magnesium sulphate should be considered for patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy.

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Pharmacology: Respiratory and Endocrine

Question 71 of 76

Regarding the management of acute asthma in adults, which of the following statements is INCORRECT:

a)
Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care. ✗
b)
Parenteral hydrocortisone or intramuscular methylprednisolone are alternatives in patients who are unable to take oral prednisolone.
c)
Intravenous magnesium sulphate should be considered for patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy.
d) High-dose inhaled short-acting beta2-agonists are the first line treatment for acute asthma.
e)
Nebulised magnesium sulphate should be considered for patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy. ✓

Answer

The BTS/SIGN guidelines state that nebulised magnesium is not recommended for treatment in adults with acute asthma but that a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) should be considered in patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2< 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

Question 72 of 76

Prednisolone would be most useful for which of the following conditions:

a Topical use in eczema

b Cerebral oedema secondary to malignancy

c Mineralocorticoid replacement in Addison's disease

d Long-term suppression in polymyalgia rheumatica

e Emergency management of anaphylaxis

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Pharmacology: Respiratory and Endocrine



Question 72 of 76

Prednisolone would be most useful for which of the following conditions:

a) **Topical use in eczema** ✖
b) Cerebral oedema secondary to malignancy
c) Mineralocorticoid replacement in Addison's disease
d) Long-term suppression in polymyalgia rheumatica ✔
e) Emergency management of anaphylaxis

Answer

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Notes

In comparing the relative potencies of corticosteroids in terms of their anti-inflammatory effects, it is worth noting that high glucocorticoid activity in itself is of no advantage unless it is accompanied by relatively low mineralocorticoid activity.

Anti-inflammatory potency

Equivalent anti-inflammatory doses of corticosteroids:

Prednisolone 5 mg = Dexamethasone 750 micrograms = Hydrocortisone 20 mg

This does not take into account their mineralocorticoid effects, nor their duration of action.

Side effects

Overdosage or prolonged use can exaggerate some of the normal physiological actions of corticosteroids leading to mineralocorticoid and glucocorticoid side effects. Mineralocorticoid side effects are most marked with fludrocortisone, but are significant with hydrocortisone, negligible with the high potency glucocorticoids, betamethasone and dexamethasone, and occur only slightly with methylprednisolone and prednisolone.

Mineralocorticoid side effects include:

- hypertension
- sodium retention
- water retention and oedema
- potassium loss
- calcium loss

Glucocorticoid side effects include:

- weight gain
- hyperglycaemia and diabetes
- osteoporosis and osteoporotic fractures
- muscle wasting (proximal myopathy)
- peptic ulceration and perforation
- psychiatric reactions

Side effects can be minimised by using the lowest effective dose for the minimum period possible. The suppressive action of a corticosteroid on cortisol secretion is least when it is given as a single dose in the morning. Whenever possible local treatment with creams, intra-articular injections, inhalations, eye drops, or enemas should be used in preference to systemic treatment.

Adrenal suppression

During prolonged therapy with corticosteroids, particularly with systemic use, adrenal atrophy develops and can persist for years after stopping. Abrupt withdrawal after a prolonged period can lead to acute adrenal insufficiency, hypotension, or death. Patients removed from long-term glucocorticoid therapy must be weaned off the drug over several days, using progressively lower doses to allow recovery of adrenal responsiveness.

To compensate for a diminished adrenocortical response caused by prolonged corticosteroid treatment, any significant intercurrent illness, trauma, or surgical procedure requires a temporary increase in corticosteroid dose, or if already stopped, a temporary reintroduction of corticosteroid treatment.

Patients on long-term corticosteroid treatment should carry a steroid treatment card which gives guidance on minimising risk and provides details of prescriber, drug, dosage and duration of treatment.

Infections

Prolonged courses of corticosteroids increase susceptibility to infections and severity of infections; clinical presentation of infections may also be atypical.

Unless they have had chickenpox, patients receiving oral or parenteral corticosteroids for purposes other than replacement should be regarded as being at risk of severe chickenpox. Manifestations of fulminant illness include pneumonia, hepatitis and disseminated intravascular coagulation; rash is not necessarily a prominent feature. Passive immunisation with varicella–zoster immunoglobulin is needed for exposed non–immune patients receiving systemic corticosteroids or for those who have used them within the previous 3 months. Confirmed chickenpox warrants specialist care and urgent treatment. Corticosteroids should not be stopped and dosage may need to be increased.

Patients taking corticosteroids should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

Hydrocortisone

The relatively high mineralocorticoid activity of hydrocortisone, and the resulting fluid retention, makes it unsuitable for disease suppression on a long-term basis. However, hydrocortisone can be used for adrenal replacement therapy (together with fludrocortisone). Hydrocortisone is primarily used on a short-term basis by intravenous injection for the emergency management of some conditions such as acute asthma and allergic/anaphylactic reactions as an adjunct to adrenaline. The relatively moderate anti-inflammatory potency of hydrocortisone also makes it a useful topical corticosteroid for the management of inflammatory skin conditions because side effects (both topical and systemic) are less marked. Hydrocortisone acetate or one of the synthetic analogues is also generally used for local injection.

Prednisolone

Prednisolone, an intermediate-acting agent, has predominantly glucocorticoid activity with only minimal mineralocorticoid activity. Prednisolone is the corticosteroid most commonly used by mouth for long-term disease suppression, for example in rheumatoid arthritis and temporal arteritis.

Dexamethasone

Dexamethasone has a very high glucocorticoid activity in conjunction with insignificant mineralocorticoid activity. This makes it particularly suitable for high-dose therapy in conditions where fluid retention would be a disadvantage such as in the management of raised intracranial pressure or cerebral oedema secondary to malignancy. Dexamethasone also has a long duration of action and this, coupled with its lack of mineralocorticoid action makes it particularly suitable for suppression of corticotropin secretion in congenital adrenal hyperplasia. In most individuals a single dose of dexamethasone at night, is sufficient to inhibit corticotropin secretion for 24 hours. This is the basis of the 'overnight dexamethasone suppression test' for diagnosing Cushing's syndrome.

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Pharmacology: Respiratory and Endocrine

Question 73 of 76

Which of the following clinical features is a cardinal sign of near-fatal asthma:

a Altered conscious level

b Silent chest

c PEFR < 33% of best or predicted

d $PaCO_2$ > 6.0 kPa

e PaO_2 < 8.0 kPa

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Pharmacology: Respiratory and Endocrine

Question 73 of 76

Which of the following clinical features is a cardinal sign of near-fatal asthma:

a) Altered conscious level ✖
b) Silent chest
c) PEFR < 33% of best or predicted
d) PaCO2 > 6.0 kPa ✔
e) PaO2 < 8.0 kPa

Answer

The BTS/SIGN guidelines defines near-fatal asthma as a raised PaCO2 and/or requiring mechanical ventilation with raised inflation pressures.

Notes

Asthma is a common chronic inflammatory condition of the airways characterised by bronchoconstriction. The most frequent symptoms are cough, wheezing, chest tightness, and shortness of breath. The bronchoconstriction is usually reversible (either spontaneously or with the aid of medication) leading to intermittent symptoms, but in some patients with chronic asthma the inflammation may result in irreversible airway obstruction. Occasionally, asthma symptoms can get gradually or suddenly worse provoking an acute asthma attack that, if severe, may require hospitalisation.

Classification of acute asthma in adults

The nature of treatment required for the management of acute asthma depends on the level of severity, described as follows:

Severity	Criteria
Moderate	• Increasing symptoms • PEFR > 50 – 75% of best or predicted • No features of acute severe asthma
Severe	Any one of: • PEFR 33 – 50% of best or predicted • Respiratory rate ≥ 25 breaths/minute • Heart rate ≥ 110 beats/minute • Inability to complete sentences in one breath
Life-threatening	Any one of the following in someone with severe asthma: • PEFR < 33% of best or predicted • SpO2 < 92% or PaO2 < 8 kPa • Normal PaCO2 (4.6 – 6.0 kPa) • Silent chest • Cyanosis • Poor respiratory effort • Arrhythmia • Hypotension • Exhaustion • Altered conscious level
Near-fatal	Respiratory acidosis (increased arterial PaCO2) and/or requiring mechanical ventilation with increased inflation pressures

BTS/SIGN management of acute asthma in adults

Patients with moderate asthma should be treated at home or in primary care according to response to treatment, while patients with severe or life-threatening acute asthma should start treatment as soon as possible and be admitted to hospital immediately following initial assessment.

Management of acute asthma:

- **Oxygen therapy:**
  - Give controlled supplementary oxygen therapy for all hypoxaemic patients with severe acute asthma to maintain saturations of 94 – 98% (unless patient at risk of hypercapnic respiratory failure)
- **High-dose inhaled short-acting beta2-agonists** are the first line treatment (salbutamol or terbutaline):
  - A pressurised metered dose inhaler with spacer device is preferred in patients with moderate to severe asthma (4 puffs initially, followed by 2 puffs every 2 minutes according to response, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary)
  - The oxygen-driven nebulised route is recommended for patients with life-threatening features or poorly responsive severe asthma (salbutamol 5 mg at 15 – 30 minute intervals)
  - Consider continuous nebulisation in patients with severe acute asthma that is poorly responsive to initial bolus dose (salbutamol at 5 – 10 mg/hour)
  - Reserve intravenous route for those in whom inhaled therapy cannot be used reliably
- **Ipratropium bromide (muscarinic antagonist):**
  - Add nebulised ipratropium bromide (0.5 mg 4 – 6 hourly) to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy to provide greater bronchodilation
- **Steroid therapy:**
- Steroids reduce mortality, relapses, subsequent hospital admission and requirement for β2 agonist therapy. The earlier they are given in the acute attack the better the outcome.
  - Give steroids in adequate doses (prednisolone 40 – 50 mg) to all patients with an acute asthma attack; steroid tablets are as effective as injected steroids, provided they can be swallowed and retained
  - Continue oral prednisolone (40 – 50 mg) daily for at least five days or until recovery (steroids can usually be stopped abruptly, dose does not need tapering)
  - Parenteral hydrocortisone (100 mg every 6 h, 400 mg daily) or intramuscular methylprednisolone (160 mg) are alternatives in patients who are unable to take oral prednisolone
- **Magnesium sulphate:**
  - Nebulised magnesium is not recommended for treatment in adults with acute asthma
  - Consider giving a single dose of IV magnesium sulphate (1.2 – 2 g IV infusion over 20 minutes) to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- **Aminophylline:**
  - Intravenous aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroids and may cause side effects such as arrhythmias and vomiting.
  - However, some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr)
- Intravenous magnesium sulphate or aminophylline should only be used following consultation with senior medical staff
- **Routine prescription of antibiotics is not indicated** for patients with acute asthma

Drug	Dose
Oxygen	Give controlled supplementary oxygen therapy for all hypoxaemic patients with acute severe asthma to maintain saturations of 94 – 98%
Salbutamol	• pMDI + spacer (moderate/severe asthma): 4 puffs initially, followed by 2 puffs every 2 minutes, up to 10 puffs, whole process repeated every 10 – 20 minutes if necessary • Oxygen-driven nebuliser (life-threatening or poorly responsive severe asthma): salbutamol 5 mg at 15 – 30 minute intervals • Continuous nebulisation (asthma that is poorly responsive to initial bolus dose): salbutamol at 5 – 10 mg/hour • Reserve intravenous route in those in whom inhaled therapy cannot be used reliably
Ipratropium bromide	Add nebulised ipratropium bromide 0.5 mg 4 – 6 hourly to nebulised beta2-agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to beta2-agonist therapy
Steroid	• Oral prednisolone 40 – 50 mg daily for 5 days (or until recovery) • Intravenous hydrocortisone 100 mg every 6 h (400 mg daily) until patients can take oral therapy • Intramuscular methylprednisolone 160 mg in patients unable to take oral therapy
Magnesium sulphate	• Nebulised magnesium is not recommended for treatment in adults with acute asthma • Consider giving a single dose of IV magnesium sulphate 1.2 – 2 g IV infusion over 20 minutes to patients with acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
Aminophylline	Some patients with near-fatal asthma or life-threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline 5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then continuous infusion of 0.5 – 0.7 mg/kg/hr
Antibiotics	Not routinely prescribed in acute asthma

Patient disposal

Patients whose PEFR is > 75% of predicted or best one hour after initial treatment may be discharged from ED (unless there are other reasons why admission is appropriate). It is essential that the patient's primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.

Admit all patients:

- with any features of life-threatening or near-fatal asthma attack
- with any feature of severe asthma attack persisting after initial treatment

Refer to ITU any patient:

- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing decreased pH or increased H+
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2016/

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Pharmacology: Respiratory and Endocrine

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Glucagon may be used as an antidote for overdose with which of the following:

a Calcium channel blockers

b Beta blockers

c SSRIs

d Theophylline

e Digoxin

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Pharmacology: Respiratory and Endocrine

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Glucagon may be used as an antidote for overdose with which of the following:

a) Calcium channel blockers
b) **Beta blockers** ✔
c) SSRIs
d) Theophylline
e) Digoxin

Answer

Glucagon is mainly used as a treatment for hypoglycaemia but may also be used as an antidote in beta-blocker overdose and in anaphylaxis in patients on beta-blockers that fail to respond to adrenaline.

Notes

Management of hypoglycaemia in adults

- In adults who are conscious, cooperative and can swallow:
  - Give 15 – 20 g quick acting carbohydrate of the patient's choice where possible e.g. 90 – 120 mL of Lucozade or 5 – 7 Dextrosol tablets
  - Repeat capillary blood glucose 10 – 15 minutes later
  - If blood glucose is still < 4.0 mmol/L, repeat step 1 (no more than 3 treatments in total)
  - If blood glucose remains < 4.0 mmol/L after 45 minutes or 3 cycles, consider:
    - 1 mg glucagon IM
    - IV 10% glucose infusion at 100ml/hr
- In adults who are conscious but uncooperative:
  - Give either 1.5 – 2 tubes Glucogel/Dextrogel (may repeat up to 3 times)
  - If this is ineffective give glucagon 1 mg IM (may only give once)
  - If blood glucose level remains less than 4.0 mmol/L after 45 minutes (or 3 cycles), consider IV 10% glucose infusion at 100ml/hr
- In adults who are unconscious:
  - Give either:
    - 1 mg glucagon intramuscularly (if not effective after 10 – 15 minutes, IV glucose should be given)
    - 75 – 80 ml of 20% glucose intravenously over 10 – 15 minutes
    - 150 – 160 ml of 10% glucose intravenously over 10 – 15 minutes

Once blood glucose is > 4.0 mmol/L and the patient recovered, give a long acting carbohydrate of the patient's choice where possible e.g. two biscuits, one slice of bread. Note that patients given glucagon require a larger portion of long acting carbohydrate to replenish glycogen stores (double the suggested amount).

If the hypoglycaemia was due to sulfonylurea or long acting insulin therapy then be aware that the risk of hypoglycaemia may persist for up to 24 – 36 hours following the last dose, especially if there is concurrent renal impairment.

Glucagon

Glucagon, a polypeptide hormone produced by the alpha cells of the islets of Langerhans, increases plasma-glucose by mobilising glycogen stored in the liver. Glycogen promotes glycogenolysis and gluconeogenesis.

Glucagon may take up to 15 minutes to have an effect and will be less effective in alcoholics, prolonged starvation and severe liver disease when glycogen stores are depleted. In this situation or if prolonged treatment is required, IV glucose is better. Glucagon may also be less effective in patients prescribed sulfonylurea therapy.

Glucagon may also be used as an antidote in beta-blocker overdose and in anaphylaxis in patients on beta-blockers that fail to respond to adrenaline.

Glucagon is contraindicated in pheochromocytoma.

Adverse effects include:

- Nausea and vomiting
- Hyperglycaemia
- Hypokalaemia
- Hypotension

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Pharmacology: Respiratory and Endocrine

Question 75 of 76

Which of the following is NOT a typical side effect of salbutamol:

a QT-interval prolongation

b Headache

c Hyperkalaemia

d Arrhythmias

e Tremor particularly affecting the hands

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Pharmacology: Respiratory and Endocrine



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Which of the following is NOT a typical side effect of salbutamol:

a) QT-interval prolongation
b) Headache
c) **Hyperkalaemia** ✔
d) Arrhythmias
e) Tremor particularly affecting the hands

Answer

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
- Hypokalaemia
- Cardiac arrhythmia and paradoxical bronchospasm (rare)
- Acute angle-closure glaucoma
- QT-interval prolongation

Notes

Selective beta-2 agonists act directly on beta-2 receptors, causing smooth muscle relaxation and dilation of the airways.

Mild to moderate symptoms of asthma respond rapidly to the inhalation of a selective short-acting beta2 agonist such as salbutamol or terbutaline sulfate. Short-acting beta-2 agonists have a rapid onset of action (15 minutes) and their effects last for up to 4 hours. Salbutamol or terbutaline sulfate can be given intravenously for severe or life-threatening acute asthma; patients should be carefully monitored and the dose adjusted according to response and heart rate.

Short acting beta-2 agonists are used for immediate relief of asthma symptoms, while some long-acting β2 agonists (e.g. salmeterol) are added to an inhaled corticosteroid in patients requiring prophylactic treatment.

Cautions

Beta-2 agonists should be used with caution in people with:

- **Cardiovascular disease** including arrhythmias and hypertension (beta-2 agonists may cause an increased risk of arrhythmias and significant changes to blood pressure and heart rate)
- **Diabetes** (risk of hyperglycaemia and ketoacidosis, especially with intravenous use)
- **Hyperthyroidism** (beta-2 agonists may stimulate thyroid activity)
- **Hypokalaemia** (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline, corticosteroids, diuretics and by hypoxia)
- **Susceptibility to QT-interval prolongation**
- **Convulsive disorders**

Interactions

Hypokalaemia may be potentiated by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics. This in turn may predispose to toxicity in patients taking digoxin.

Side effects

Side effects are usually dose related and include:

- Fine tremor — occurs particularly in the hands and is usually worse in the first few days of treatment.
- Palpitations and tachycardia
- Headache
- Seizure
- Anxiety
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Pharmacology: Respiratory and Endocrine

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What is the mechanism of action of theophylline:

a Beta-2 agonist

b Muscarinic antagonist

c Phosphodiesterase inhibitor

d Acetylcholinesterase inhibitor

e Dopamine antagonist

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What is the mechanism of action of theophylline:

a) Beta-2 agonist ✗
b) Muscarinic antagonist
c) Phosphodiesterase inhibitor ✓
d) Acetylcholinesterase inhibitor
e) Dopamine antagonist

Answer

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Notes

Theophylline may have an additive bronchodilation effect when used in conjunction with small doses of beta-2 agonists. Theophylline is a xanthine which inhibits phosphodiesterase resulting in increased tissue concentrations of cyclic adenosine monophosphate (cAMP).

Theophylline is metabolised in the liver, and has a narrow therapeutic index. The plasma theophylline concentration is increased in heart failure, hepatic impairment, in viral infections, in fever and in the elderly. A reduction in dosage may be necessary to avoid toxic accumulation. The plasma theophylline concentration is decreased in smokers, and by alcohol consumption.

Indications

It is used as a bronchodilator in asthma and stable COPD. It is not generally effective in exacerbations of chronic obstructive pulmonary disease, but is used rarely for severe or life-threatening acute asthma given by injection as aminophylline, a mixture of theophylline with ethylenediamine, which is 20 times more soluble than theophylline alone.

Contraindications

Theophylline should not be prescribed to:

- People with porphyria
- People with hypersensitivity to xanthines
- Children concomitantly receiving ephedrine
- Infants under 6 months of age

Cautions

Theophylline should be used with caution in people with:

- Cardiac arrhythmias or other cardiac disease
- Hepatic impairment
- Epilepsy
- Hypertension
- Hyperthyroidism
- Peptic ulcer
- Risk of hypokalaemia

Interactions

Hypokalaemia may be potentiated by concomitant therapy with beta-2 agonists, corticosteroids and diuretics.

Excretion of lithium may be potentiated by concomitant therapy with theophylline.

There is an increased risk of convulsions when theophylline is given with quinolones.

Examples of enzyme-inhibiting drugs (raise plasma theophylline level)	Examples of enzyme-inducing drugs (lower plasma theophylline level)
• Erythromycin • Clarithromycin • Ciprofloxacin • Fluconazole • Verapamil • Allopurinol • Cimetidine	• Primidone • Phenobarbital • Carbamazepine • Phenytoin • Ritonavir • Rifampicin • St John's Wort

Side effects

Side effects include:

- Tachycardia, palpitations and arrhythmias
- CNS stimulation, tremor, headache, insomnia and convulsions
- Gastric irritation, nausea, vomiting and diarrhoea
- Hypokalaemia (potentially serious hypokalaemia may result from beta-2 agonist therapy; this effect may be potentiated in severe asthma by concomitant treatment with theophylline and its derivatives, corticosteroids, and diuretics, and by hypoxia)

Monitoring requirements

In most individuals, a plasma-theophylline concentration of 10 – 20 mg/litre (55 – 110 micromol/litre) is required for satisfactory bronchodilation, although a lower plasma-theophylline concentration of 5 – 15 mg/litre may be effective. Adverse effects can occur within the range 10 – 20 mg/litre and both the frequency and severity increase at concentrations above 20 mg/litre.

Plasma-theophylline concentration is measured 5 days after starting oral treatment and at least 3 days after any dose adjustment.

Overdose

Theophylline in overdose can cause vomiting (which may be severe and intractable), agitation, restlessness, dilated pupils, sinus tachycardia, and hyperglycaemia. More serious effects are haematemesis, convulsions, and supraventricular and ventricular arrhythmias. Severe hypokalaemia may develop rapidly.

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